Texas; December, 19

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TEXAS STATE JOURNAL OF MEDICINE

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THE GRIEVANCE COMMITTEE

The problem of professional self-discipline within the medical profession has always been difficult and has been highlighted among criticisms directed toward the medical profession in the past several years. Several state medical associations have attempted to remedy this situation by providing for a statewide committee to hear and investigate complaints made against doctors by the public, thus augmenting the program of self-discipline of the county medical societies and assisting the boards of censors of these societies. Such a state committee acts more or less as a grand jury whose function it is to investigate, advise, and prosecute if necessary before the proper judicial body, be it county medical society through the board of censors, the board of councilors of the state association, the board of medical examiners, or a civil court in extreme cases in which such action is deemed justifiable.

The first state medical association to develop such a plan, so far as we are able to discover, was the Colorado State Medical Society. Realizing the need for some more effective means of answering the criticism of the public relative to alleged overcharging and negligence of patients and at times charges of malpractice, the Colorado society amended its constitution and by-laws to provide for a Board of Supervisors consisting of twelve members of the society, none of whom hold any other office in the state or county societies of which they are members, and with no two members of the board from the same county medical society. This "grand jury" serves in a completely impartial manner made increasingly so by the fact that any member of the board who is a member of the same county society as the doctor under investigation becomes disqualified for any participation on the board in that particular case.

The functions of the Board of Supervisors are as follows:

1. To receive complaints from anyone—doctors, laymen, hospitals, organizations, medical society staff members—about any dissatisfaction in the medical field.

2. To investigate facts in the case.

3. To attempt to mediate the dispute and effect a settlement which is mutually satisfactory to complainant and defendant.

4. To prosecute the case before a proper judicial body if necessary.

5. To carry on a continuous intra-society educational program on medical ethics and patient relations, primarily through personal appearances of board members at county society meetings.

A somewhat similar plan has been established by the Oklahoma State Medical Association in the past year. The group within the state association to which complaints are referred is called the Grievance Committee and consists of the five immediate living past presidents of the association. The objectives and methods of procedure are similar if not the same as those of the Colorado plan, as is true with the plans of the four or more other state medical associations which have followed the lead of Colorado.

The fact that the names of the members of the Grievance Committee are known to the public and that these men are held in high regard and have the confidence of both public and profession because of the lofty position they have previously held in their medical organization produces a feeling of assurance that impartial justice will be had.

The Colorado plan, which has been in operation for two years, and the more recently established Oklahoma plan both have received high praise through editorial comments in the press and by members of the laity and members of the medical profession. Apparently both of these plans have been eminently successful thus far and neither board nor committee has encountered any difficulty it could not cope with satisfactorily.

The majority of complaints received have been due to misunderstandings which have been easily settled by bringing the doctor and the patient together to talk over the difficulty. Often these misunderstandings have been due to failure on the part of the doctor to explain the nature of his charges; the place of laboratory, roentgen-ray, and hospital charges; charges of the anesthesiologists; the cost of drugs; and so forth, which enter into the total cost of a specific illness. It is pointed out that by a little more diligence on the part of the doctors many of these difficulties can be avoided. Some of the types of complaints other than those arising from a belief on the part of the patient that he has been overcharged are the following: unjustified complaints of psychopathic persons or of "dead beats," ungentlemanly conduct of doctors in public, undue familiarity with female patients, public scandals, criminal abortions, unfavorable results from supposedly wrong diagnosis, performance of unjustified surgery, refusal to answer emergency calls, neglect of patients, and malpractice of different kinds.

At the recent clinical session of the American Medical Association held at Washington, D. C., the House of Delegates commended those states that have grievance committees and recommended that all state medical associations which have not already done so investigate the advisability of forming such committees. The Board of Trustees of the State Medical Association of Texas has had such a plan under advisement for some time and has referred the matter to the Committee on Public Relations for study. No doubt a report will be forthcoming in the near future.

NARCOTICS PRESCRIPTIONS

A problem of concern to physicians and druggists throughout the nation was brought before the United States District Court for the Eastern District of Michigan, Southern Division, by the report of a grand jury on June 30, 1949. This problem concerns the dispensing of narcotics without properly executed prescriptions.

The grand jury concluded that the "vicious" practice of pharmacists dispensing narcotics upon telephone orders from physicians is rather general. It was pointed out that federal law

requires the filing of a written prescription signed by a physician to make the sale of narcotics legal. While the necessity of permitting occasional sales upon telephoned authorization in certain emergencies was recognized, the grand jury declared that the practice is much too common and that written prescriptions confirming the telephoned orders are frequently overlooked. It was explained that such a practice presents opportunities for the diversion of narcotics through illicit channels and frustrates the whole system of regulation and control of narcotics.

The testimony presented before the grand jury in Detroit indicated that the practice referred to is prevalent throughout the United States, and the jury stated that in its opinion a "comprehensive investigation" is warranted in other federal districts.

Whether or not investigations by federal authorities appear to be necessary in Texas, the problem raised in Michigan should not be treated lightly. As pointed out by the grand jury, physicians sometimes have failed to realize "the importance and dignity of a prescription, its function and value." Prescriptions in California have been considered of sufficient importance that official prescription forms in triplicate are issued by a state agency. The physician furnishes one of the forms to the patient, another is sent to the state, and the third is kept by the doctor. Forgeries have been reduced to a minimum; the physicians exercise more care against the loss or theft of the prescription forms; the investigative burdens of the Bureau of Narcotics have been lessened; and the regulations concerning the delivery of the prescription to the pharmacist by the doctor has enjoyed a greater compliance. The grand jury suggested that the method used by California is a good one to be followed.

Certainly physicians should recognize that "any doctor requesting a druggist to dispense narcotics without the production of a signed prescription, except in an emergency situation, is equally guilty with the druggist in violating the law." A prescription, plainly written and signed with a full signature, should be supplied by the physician for each order of narcotics as a protection to himself, his patient, the pharmacist, and the system of narcotics regulation.

THE CHRISTMAS SEAL

The tuberculosis seal is now a part of Christmas, but until 1904 there were no such seals to bring a happy holiday greeting and the hope of better health in the coming year.

Einar Holboell, a postal clerk in Copenhagen, Denmark, who loved children, thought as he sorted mail of what a fine sum could be raised to help sick children if each letter or package bore another stamp, sold to aid the fund. His idea was approved by King Christian, and more than 4,000,000 stamps were sold in Copenhagen during the Christmas season of 1904.

In July, 1907, an article in the *Outlook* by Jacob Riis, a Danish-born American, urged the adoption of the seals in this country to fight tuberculosis. Later that year Miss Emily Bissell, a public health worker in Wilmington, Del., became concerned about the financial difficulties of a small sanatorium and remembered Riis's plea. With the aid of a few friends Miss Bissell launched the first Christmas stamp sale in America on December 9, 1907. She and Leigh Mitchell Hodges, a columnist on the Philadelphia *North American* who had become interested in the project, persuaded the American Red Cross to sponsor a nationwide Christmas seal sale the next year. From 1910 to 1919 the National Tuberculosis Association cooperated with the Red Cross in the sales. In 1919 the red cross on the seals was changed to the double-barred cross of Lorraine, symbol

of the Tuberculosis Association, and in 1920 the Tuberculosis Association became the sole sponsor of the seals. In Texas alone, $664,493.68 was obtained from seal sales in 1948. Of this sum, 95 per cent remained in Texas and 5 per cent went to the National Tuberculosis Association for its educational and research program.

Although revenue from the seals has done much through the years to reduce the toll which tuberculosis has exacted in incapacitating illness and useless death, tuberculosis still kills more people between the ages of 15 and 34 than any other disease. Physicians who may not have bought tuberculosis Christmas seals this year will certainly want to do so before the holiday season is over, remembering the help which their sale has brought to the cause of better health since Einar Holboell thought of them forty-five years ago.

AMERICAN MEDICAL ASSOCIATION DUES

The House of Delegates of the American Medical Association, meeting December 6-8 in Washington, adopted dues of $25 for each active member of the Association for 1950. Details of this action are not available as this JOURNAL goes to press, but Dr. George F. Lull, Chicago, Secretary and General Manager of the A.M.A., has indicated that the dues will be collected by state and county societies in accordance with local custom and then will be transmitted to the A.M.A.

It should be pointed out that the new dues are for membership, not fellowship, in the A.M.A. Heretofore each member of a county medical society has been a member of the A.M.A. through his state medical association. Until the recent action of the House of Delegates, membership in the A.M.A. had been effective without payment of dues to the national organization, although during the past year members were expected to pay an assessment to help carry on the work of the organization.

Members of the State Medical Association of Texas may expect to receive a letter soon indicating how the A.M.A. dues should be paid and from whom they will be expected. It seems to be the intent of the House of Delegates that intern and resident, honorary, emeritus, and military members need not pay, but the point has not been clarified. Secretaries of county medical societies may continue to forward to the State Secretary such State dues as have already been collected without reference to the new A.M.A. dues, but the $25 A.M.A. dues from those members who wish to include them may be sent in with the State dues.

ANOTHER VOLUME OF JOURNAL CLOSED

This number of the TEXAS STATE JOURNAL OF MEDICINE completes the first volume of the publication since the cover, type style, and make-up were changed. These changes have created considerable comment, uniformly favorable, and resulted in an award for improvement presented by the Society of Associated Industrial Editors.

Volume 45 contained 1,722 pages, including 830 pages of advertising and 892 pages of reading matter. There were 81½ pages of editorials, 393½ of original articles and case reports, and 417 of miscellaneous news, library items, organization material, Woman's Auxiliary information, and death notices. Ninety-two original articles and case reports were published.

During the coming year, the Editors hope to learn through a special questionnaire what readers of the JOURNAL do and do not like about its contents and make-up. The results of the survey will be considered in further efforts to enhance the value of the periodical. Meanwhile, comments and suggestions from readers will be welcome.

CURRENT EDITORIAL COMMENT

THE ROAD AHEAD

A small book recently published, "The Road Ahead"* by John T. Flynn,† paints a dramatic picture of England's march toward its present socialistic state and draws a parallel with what is now happening in the United States. This book offers assistance to anyone who wishes to understand the changing social and political scene.

Mr. Flynn presents clearly how England was invaded with the socialistic idea in what was a most insidious manner. It is peculiar to think that an organization like the Fabian Society, which was organized in England in 1883, according to Mr. Flynn, could work so effectively toward the socialization of all England and completely avoid the use of the word "socialism" and in addition keep the English people from knowing they were actually being socialized! It is apparent from this book that with all his ability, Lloyd George was completely without understanding as to the Party's intention. Through the capture of the Labor Party and the Liberal Party it did not take the Socialists too long to dominate completely the thinking of the English Parliament. Mr. Flynn says, "Thus only 18 years after the start, literally from scratch, of the Socialist drive, their leader was England's Prime Minister. Instead of cutting the claws of the Labor government, Asquith had cut the throat of the Liberal Party. Asquith's Liberals and Stanley Baldwin's Conservative voters represented a vast majority of the people. But Asquith turned the machinery of the British Empire over to the Socialist Labor Party representing only a third of the electorate and with only 191 votes out of 615 seats in the Commons."

It is interesting to see how closely the pattern in America is following the pattern in England. The distinction which Mr. Flynn makes between Communism in Russia and Socialism in England leaves no doubt in the mind of the reader that they are essentially one and the same thing, the only difference being in the policy of attack of the two organizations. Russian Communism is a type of socialism which seeks victory by a blow on the head, while the English type, he says, is a Fabianistic policy which is giving America more trouble now than the Communistic policy. He says further that we are fighting vigorously a few hundred thousand Communists of the Russian socialistic philosophy and embracing the socialistic policy that is taught and promulgated by England.

The extent to which socialism has crept into our society is suggested in a chapter entitled "The 'Kingdom of God,' " which no one, Christian or otherwise, can afford to overlook.

In a recent syndicated column, Westbrook Pegler said: "On second reading, I sincerely believe that John T. Flynn's new book . . . called 'The Road Ahead—America's Creeping Revolution,' is one of the greatest political pamphlets in our history. Flynn awakens me to the realization that Socialism is Communism and that therefore the 'planners,' as our Socialists call themselves for disguise, are promoting the preparatory phases of Communism. . . . I earnestly urge you to read it carefully . . . and be warned."

For those concerned with reversing the direction in which he believes America is going —and this should include every doctor and all other citizens as well—Mr. Flynn suggests ten bases for action:

This department of the JOURNAL *presents editorial comments on current items pertaining to the science, art, and practice of medicine, contributed by members of the State Medical Association and scientists closely associated with the medical profession of Texas. Invitation is hereby extended to any member of the State Medical Association of Texas to submit such discussions for this department. The discussions should not be more than 500 words in length.*

**Published by the Devin-Adair Company, New York, 1949. Available from the publisher in a cloth bound edition at $2.50 per copy and from the Committee for Constitutional Government, Inc., 205 East Forty-Second Street, New York 17, in a special paper bound edition at 50 cents per copy.*

†Editor, lecturer, economist, educator, and author of "The Roosevelt Myth" and other books.

1. We must put human freedom as the first of our demands.

2. We must stop apologizing for our capitalistic society.

3. We must not take one more step into socialism.

4. We must get rid of the compromising leaders.

5. We must recognize that we are in the midst of a revolution and begin to fight it as such.

6. We must put an end to the orgy of spending.

7. We must put an end to crisis government.

8. We must stop "planning" for socialism and begin planning to make our free system of private enterprise operate at its highest capacity.

9. We must set about rebuilding in its integrity our republican system of government.

10. We cannot depend on any political party to save us. We must build a power outside the parties so strong that the parties will be compelled to yield to its demand.

WILLIAM M. GAMBRELL, M. D., President-Elect,
State Medical Association of Texas,
Austin, Texas.

Capital National Bank Building.

CONTROLLING TUBERCULOSIS IN COLLEGE STUDENTS

The rate of tuberculosis reported from surveys of college students in Texas is constant at 0.6 per cent. In a student body of 10,000, about 60 will show disease. Perhaps 12 or 15 will require treatment in a sanatorium; the balance will require close medical supervision to avoid danger of contagion and further advancement of the disease. While these figures remain constant, school authorities must conduct annual surveys of some type or accept the responsibility for exposure of large numbers of susceptible subjects to open cases.

Since November, 1946, when the Texas Tuberculosis Association conducted the first microfilm survey among students at the University of Texas, about thirty-five Texas colleges have had similar campaigns. There are now records of more than 60,000 such chest examinations.

Some American colleges require annual tuberculin skin tests of all students and make chest films of only positive reactors. Some years ago when 40 to 50 per cent of student bodies reacted positively, the cost of chest films was considerable, but in recent years with rates reported as low as 10 and 15 per cent, the cost, even when the large film is used, is small. Although a microfilm survey can be done at less cost than skin testing, the informational and educational value of the skin test in a college control program more than makes up for the difference.

Two important facts in the tuberculosis picture may soon have to be reckoned with: (1) the annual decrease in the tuberculous death rate and (2) the aging of the tuberculous population.

The rate of death from tuberculosis in Texas fell from 68.6 to 38.6 per 100,000 population or 44 per cent in the period from 1937 to 1947. If this can mean the gradual elimination of the disease, a time may come when surveys will not be worth the cost. Before the survey is discarded, however, the aging tuberculous population must be considered.

In 1900, 64 per cent of all deaths from tuberculosis were in the 15 to 45 age group and only 24 per cent in the ages beyond. By 1948 only 30.6 per cent died in the 15 to 45 age period and 60.5 per cent in the older age period. As the disease takes refuge in the older age groups we are apt to be deceived by mortality figures and think the enemy is being routed faster than is actually true. Large numbers of deaths in this older age group will be

finally recorded as caused by coronary occlusion, cerebral hemorrhage, or even accident without any notation of the tuberculous infection that may have been present.

Although the rate is not high in college students, they will shortly be the parents of small children, highly susceptible to the infection. The most effective way to reduce the acid-fast storage in the ever increasing aged population is to reduce the number of young people who are allowed to acquire and carry the infection into that age period. When we consider also that treatment can offer good prospects for cure only in early stages, it must be concluded that college surveys cannot yet safely or wisely be dispensed with.

What about schools unable to set up control programs? And how can the cost of control be reduced without sacrificing efficiency? The answer to these questions and the key to the control effort generally is the private family physician. A simple plan to implement the idea would be

1. To require each student on admission to present a certificate showing a recent negative intradermal skin test or a positive test with recent chest film.

2. To require all negative reactors to repeat their tests and all positive reactors to have chest films annually.

The cost to each student would be small when compared to its value as insurance against the danger of tuberculosis that otherwise would be present. Such a college admission requirement also might do much to stimulate the lagging effort now being made to encourage high schools to adopt a plan for examining senior students for tuberculosis and thus would have great value in the control effort generally.

DR. J. EDWARD JOHNSON, Associate Physician,
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Austin, Texas.

ORIGINAL ARTICLES

THORACIC SURGERY AND THE GENERAL PRACTITIONER

JOHN S. HARTER, M.D., Louisville, Kentucky

THE rapid strides in thoracic surgery in the past ten years have made possible the rational treatment of many diseases of the chest formerly considered chronic or fatal. The general practitioner is too frequently unaware of the possibilities for cure in these diseases or may believe the cure is more dangerous than the disease. Patients are still treated for years for a disease that could be cured within two weeks and the chronic invalid completely rehabilitated. On the other hand, too many patients are still treated symptomatically for undiagnosed lesions of the chest until the possibilities for cure are lost.

Thoracic surgery is now no more dangerous than abdominal surgery. Its mortality and morbidity rates are actually lower than for gastric resection, cholecystectomy, and resection of the colon. Deformity is no longer an accompaniment of thoracic surgery. The fears of the general practitioner in referring a patient for thoracic surgery can be overcome by knowing what the patient can be offered.

Read before the Section on General Practice, State Medical Association of Texas, Annual Session, San Antonio, May 4, 1949.

DIAGNOSIS OF CHEST DISEASES

Why does the general practitioner fail to recognize early symptoms of carcinoma of the lung, bronchiectasis, lung abscess, and tuberculosis? The answer is that physical examination all too frequently does not reveal anything abnormal. Roentgenograms, if taken, are frequently misinterpreted. The patient is treated symptomatically by giving a cough mixture or analgesic, or by strapping the chest. Too often the cause of the symptoms is not determined until the lesion is well advanced.

Cough, productive or unproductive, is the most common symptom of pulmonary disease. Most patients complaining of cough have only a bronchitis, but unless they are thoroughly examined by fluoroscopy and roentgenograms of the chest, the early signs

of serious disease will be missed. The patient with a persistent cough, with or without other manifestations of disease in the chest, should be referred to the specialist for a more definitive work-up, mainly bronchoscopy and bronchograms. Carcinoma of the lung has been diagnosed by bronchoscopy in patients who had apparently normal roentgenograms of the chest.

Examination of the sputum is as important as more common laboratory examinations of the blood, urine, and feces. The stained sputum smear is typical in various inflammatory lesions of the lung such as the pneumonias, even atypical pneumonia; tuberculosis; bronchiectasis; and lung abscess. Any physician can master the technique for such examinations in a short time. Examination of the sputum for eosinophils in allergic manifestations is extremely easy. Cytologic examination of the sputum by a trained pathologist will frequently confirm a suspected diagnosis of carcinoma of the lung; however, a negative examination of the sputum for cancer cells does not rule out the diagnosis.

Hemoptysis is so alarming to patient and physician that the patient is frequently referred as soon as possible to a specialist for definitive diagnosis. Hemoptysis occurs in many conditions other than tuberculosis, such as bronchiogenic carcinoma, adenoma of the bronchus, bronchiectasis, lung abscess, broncholiths in the wall of the bronchus, mitral stenosis, hypertension, and arteriosclerosis.

Pain in the chest is practically never that of intercostal neuralgia. The cause is usually pleural involvement by (1) an inflammatory process which may be tuberculosis, pneumonia, pneumonitis, lung abscess, or an infarct or (2) direct involvement by carcinoma, either bronchiogenic or metastatic.

All that wheezes is not asthma. The relief of a wheeze by adrenaline does not always mean asthma. Foreign bodies in the larger bronchi will cause a wheeze similar to that heard in asthma. Bronchial stenosis caused by inflammatory stricture of the bronchus, bronchiogenic carcinoma, adenoma, tuberculosis, and excess mucus may cause wheezing. I have seen patients treated for more than a year for asthma because of a wheeze which resulted from a bronchiogenic carcinoma.

BRONCHIOGENIC CARCINOMA

Bronchiogenic carcinoma occurs as frequently as carcinoma of the stomach. Every practitioner will see cases of bronchiogenic carcinoma if he looks for them. The carcinoma occurs most frequently in men about 50 years of age. However, in the past year I operated on 2 patients 22 years of age for bronchiogenic carcinoma, one a man, the other a woman. The cause is not known but probably is not tobacco.

Every patient having a cough, spitting of blood, a wheeze, pain in the chest or pain radiating down the arm, pneumonia or pneumonitis localized in a lobe or lung, or a lung abscess must be suspected of having a bronchiogenic carcinoma. Pleurisy with or without fluid is frequently the initial symptom in carcinoma of the lung. Unless carcinoma of the lung is considered for every patient who displays chest symptoms, the diagnosis will not be made until late in the disease.

Loss of weight and anemia are not early symptoms of bronchiogenic carcinoma. When they occur early they are usually caused by infection following bronchial obstruction.

The average patient with bronchiogenic carcinoma waits three months before he sees his physician; he is then treated on an average of nine months before being referred to the thoracic specialist. For this reason the rate of cure at present is only about 5 per cent of those patients who have surgery. Carcinoma of the lung usually grows slowly, and if the diagnosis is made early, there is a good chance for cure.

Good roentgenograms of the chest properly interpreted are a necessity in the rational therapy of diseases of the chest. Physical examination should be used to interpret roentgenograms of the chest rather than be relied upon to determine the necessity for them. There is no roentgen-ray picture typical of bronchiogenic carcinoma. When a mass is present, the diagnosis will certainly be suspected; however, the picture may be that of pneumonia, pneumonitis, lung abscess, tuberculosis, fungus infection, atelectasis, emphysema (obstructive type), pleural effusion, and so forth, or the roentgenogram may not reveal anything abnormal.

The treatment is usually removal of the lung. A few patients in whom peripheral disease was discovered early have been treated by lobectomy. The mortality rate of pneumonectomy is extremely low in the young, increasing with the age of the patient at an average of about 10 per cent. The physiologic age of the patient is the most important factor in the mortality rate. The patient usually spends less than two weeks in the hospital, and most patients are able to carry on a normal life with one lung.

The general practitioner is the first physician to see these patients. He must recognize the possibilities of bronchiogenic carcinoma and refer the patient early if the thoracic surgeon is to increase the rate of cure.

BRONCHIECTASIS

Bronchiectasis is a dilatation of the bronchi usually occurring in people under 40. The disease usually develops in early childhood but may develop at any

time. The cause of its development in childhood is not definitely known. In some cases it is thought to be the result of neonatal atelectasis or congenital malformation. In addition bronchiectasis may develop after measles, whooping cough, or virus pneumonitis. Foreign bodies, if retained for a period of time, and lung abscesses that heal under medical management or surgical drainage may cause bronchiectasis.

There are two main types of bronchiectasis: the somewhat rare dry type, characterized by occasional hemoptysis and usually little or no cough, and the wet type, characterized by the expectoration of purulent sputum. Hemoptysis in the latter type is not common. The older patient with emphysema, cough, and expectoration practically never has bronchiectasis.

Bronchiectasis is a serious disease causing death, on an average, about eleven years after purulent expectoration is established. Patients die of a progressive pneumonitis or pneumonia. Mortality statistics do not reveal the true frequency of death due to bronchiectasis because pneumonia is usually given as the cause of death in these patients and the cause of the pneumonia, the true cause of death, is not recorded. Actually bronchiectasis is common in all sections of the country.

The diagnosis of bronchiectasis should be suspected from the history. Physical examination of the chest is frequently disappointing. When the bronchiectasis is in the base, rales can usually be heard. Apical bronchiectasis usually does not produce any abnormal signs observable upon physical examination. Roentgenograms of the chest may not reveal any abnormality or may show an infiltrative lesion in the region, which is the result of an associated pneumonitis. Apical bronchiectasis may be extremely suggestive of tuberculosis; therefore, the only definitive method of diagnosis is by the bronchogram.

Patients with the wet type of bronchiectasis may show clubbing of the fingers, but this symptom is not at all constant. Sinusitis is almost always present but is probably caused by rather than being the cause of the bronchiectasis. The sinusitis can be cured only after the bronchiectasis is cured.

Medical treatment is of little value except in preparing the patient for surgery. Postural drainage, bronchoscopic aspiration, and the antibiotics will improve the patient only temporarily. The only cure is the surgical removal of affected portions of the lung. A pneumonectomy may be necessary in some instances. In some of the bilateral cases it has been necessary to remove the middle and lower lobes of the right lung and the lower lobe and the lingula of the upper lobe of the left lung. Occasionally only portions of a lobe need be removed.

Mortality following pneumonectomy depends on the amount of lung involved, being practically nonexistent in the unilobar cases. In bilateral cases in which the disease is far advanced, the mortality rate is about 10 per cent. Patients undergoing such surgery are usually in the hospital about two weeks, and I have seen children lose only two weeks of school after a lobectomy.

LUNG ABSCESS

Lung abscess is most frequently caused by the aspiration of particulate matter, for example, during tonsillectomies or extraction of teeth. It may also be caused by infected emboli or an infarct of the lung. The organism present in a lung abscess usually causes an extremely foul sputum; however, occasionally the abscess may have a single pyogenic organism, the sputum being without odor. The putrid abscess is the result of infection caused by a combination of the fusiform bacilli, spirochetes, streptococci, staphylococci, and several other organisms.

Lung abscess is an acute infection. The patient usually runs a rather high fever and there is rapid loss of weight. Anemia develops rapidly without bleeding. Unless the infection is checked, the patient may go downhill and die in a few weeks.

Lung abscesses due to inhalation are usually single. About 50 per cent occur in the apex of the lower lobes. Abscesses caused by emboli are more apt to be multiple. Pneumonia may be followed by an abscess or the process may involve the entire lobe. Putrid empyema is an occasional complication of lung abscess, apparently the result of rupture of the abscess into the pleura. Brain abscess is another not uncommon complication of lung abscess.

The modern antibiotics have completely changed the prognosis of lung abscess. Many abscesses which are single and small, 2 to 3 cm. in diameter, will heal under medical management. If the abscess is larger, it can frequently be sterilized but will remain a cavity which will become reinfected unless resected. In some cases, particularly those with multiple cavities, it may be necessary to resect the infected portion of the lung without controlling the infection preoperatively. This is particularly true in lung abscesses developing distal to a bronchial obstruction, as in the inflammatory obstructive lesions of the middle lobe.

The medical management and preoperative regimen are essentially the same. I have been using 100,000 units of crystalline penicillin intramuscularly every three hours and .5 Gm. of streptomycin every twelve hours. Penicillin in oil and the sulfonamides have not seemed to be of much value in these mixed infections. The patients are given blood transfusions daily until the corpuscular volume is 50 per cent, and this level is maintained. The smaller

cavities frequently show signs of healing in a week and medical management is continued until the pneumonitis and cavity have disappeared. If the treatment is discontinued before the roentgen-ray picture has cleared, there will be an exacerbation. The larger and the multiple cavities are resected as soon as the patient is in condition for operation, usually after the temperature has been normal and the corpuscular volume at 50 per cent for several days and the abnormal sputum has about disappeared.

The results of resection in lung abscess have been excellent. Patients are in the hospital an average of only about three weeks and are completely well on discharge, as contrasted with the months of dressings in the cases which are drained. The mortality rate has been only about 5 per cent including the bad risk cases. Drainage is used now only for the elderly patient who cannot withstand major surgery.

TUBERCULOSIS

The thoracic surgeon has much to offer the patient who has far-advanced tuberculosis. Streptomycin has made possible the salvage of many cases formerly considered hopeless when it has been properly used with modern surgical techniques.

A word of caution about streptomycin is advisable, however. As soon as the drug became available many patients with tuberculosis, regardless of the type of lesion, were treated with it. The patients felt better and gained weight and the infiltrative lesions cleared, but unfortunately the cavities seldom healed. Even when the streptomycin was continued, the symptoms gradually recurred. Sensitivity tests revealed that the organism recovered from the patient had become streptomycin-fast. The patients had to be prepared for surgery without the protection of streptomycin and the risk of operative interference was greatly increased.

When streptomycin is used in tuberculosis, a complete cure of the lesion must be expected or the drug should be reserved for use in conjunction with surgical treatment. Tuberculous pneumonia responds well to streptomycin and miliary tuberculosis is frequently aided greatly, but cavitary tuberculosis is usually improved only slightly.

Streptomycin used with surgery has greatly decreased the period of hospitalization for the patient with pulmonary tuberculosis. Patients with minimal disease may now spend more time in the hospital than many patients with far-advanced disease. Many patients with cavities and associated acute disease can be prepared for thoracoplasty in a relatively short time by pneumoperitoneum. The patient is then given streptomycin for about a week and the thoracoplasty is started. Cavities which are too large to be collapsed by thoracoplasty are resected. In cases in which one lung is destroyed by tuberculosis and the other is relatively free of disease, a pneumonectomy can be performed. Upper-lobe lobectomies have been routinely combined with upper thoracoplasty, and total pneumonectomies with complete thoracoplasty in our clinic, the purpose being to prevent overdistention of the remaining lung, which would cause activation of any remaining foci of tuberculosis. Patients who show no roentgen-ray evidence of residual disease after resection should be given six months of bed rest to be certain that the tuberculosis is completely healed before activity is resumed.

Tuberculous cavities occurring in the lower lobe are now treated in almost every instance by primary resection. Collapse therapy has been so frequently a failure that resection seems most likely to succeed. This observation also applies to disease occurring in the middle lobe of the right lung.

EMPYEMA OF THORAX

Empyema is still one of the most mistreated diseases of the thorax. Penicillin and the sulfonamides have not changed the principles of treatment; however, they have remarkably decreased the frequency of the disease. The three most common mistakes in treatment are (1) drainage when aspiration should be done, (2) open drainage when closed drainage should be used, and (3) removal of drainage tubes before the empyema cavity is obliterated.

The rational therapy of empyema depends on identification of the organism causing the infection. The etiologic organism can be identified in most cases by a Gram or acid-fast stain.

Pneumococcus empyema should be partially aspirated until the pus becomes thick, and then open drainage should be established by rib resection and insertion of a large tube. The tube must remain in place in all empyemas that are drained until lipiodol studies show that the cavity is obliterated.

Streptococcus empyema should be treated by closed drainage to keep the lung expanded.

Staphylococcus empyema can frequently be cured by aspiration alone. Open drainage may be necessary when the cavity becomes small and aspiration is difficult.

Tuberculous empyema should not be drained but aspirated in an attempt to expand the lung and obliterate the empyema. Decortication of the lung may be necessary to obliterate the space.

MEDIASTINAL TUMORS

Tumors of the mediastinum may or may not cause symptoms but they should be removed unless such removal is contraindicated.

Mediastinal tumors cannot be accurately diagnosed preoperatively. Substernal goiters can be accurately

diagnosed in most instances. The approach to the substernal goiter is usually better from the neck because the blood supply of the goiter is inaccessible by the transthoracic approach. The potential malignancy of other mediastinal tumors, such as dermoid cysts and neurofibromas, is the indication for their removal. Unilateral lymphomas of the mediastinum may at times be removed with surprisingly long periods of apparent cure.

DISEASES OF ESOPHAGUS

Carcinoma of the esophagus can now be cured when the diagnosis is made early in the disease. The apparent rate of cure of carcinoma of the esophagus is now about the same as for carcinoma of the stomach. Too frequently patients with carcinoma of the esophagus do not present themselves for treatment early because of a lack of symptoms or because they do not realize the difficulty they are having with swallowing food or the fact that the painful swallowing may be due to cancer.

Carcinoma of the esophagus usually can be diagnosed easily by barium studies. However, it must be realized that a normal esophagus as shown by roentgen ray does not mean that cancer is not present. The diagnosis of cancer of the esophagus has been made by esophagoscopy when the esophagus appeared normal by roentgenologic studies.

The operation for cancer of the esophagus consists of resecting the esophagus well proximal to the growth, bringing the stomach up through the diaphragm and anastomosing the proximal esophagus to the stomach. Carcinoma extending to the apex of the chest may be treated in this manner because the stomach is of sufficient length when mobilized to reach to the upper portion of the chest. If the lesion cannot be resected, the obstruction can be short-circuited by intestine or stomach, allowing the patient a few months of comfort without starvation or the difficulties of a gastrostomy.

Patients with cardiospasm usually can be treated by dilatation. Some respond to large doses of thiamine chloride. If conservative therapy does not give relief, an esophagogastrostomy can be done with relative safety. I believe that patients requiring frequent dilatation are better treated by an esophagogastrostomy.

Congenital tracheo-esophageal fistulas in the newborn can be repaired. This malformation is not rare and should be kept constantly in mind as a possible cause of coughing when the newborn infant is fed or when an abnormally large amount of air is present in the intestinal tract. If feeding is continued in such a case, the child will develop a fatal pneumonia. The diagnosis can usually be made by study of the esophagus using iodized oil. If barium is used, it will enter the lungs and cause a fatal pneumonitis.

Congenital webs of the esophagus causing difficulty in swallowing in older children can be repaired.

HEART AND VASCULAR DISORDERS

Syphilitic aneurysms cannot be cured by surgery. Some aneurysms of the great vessels can be held from progression and the pain greatly relieved by wrapping the aneurysm with cellophane to cause a fibrous wall around the sac. This seems to be more logical and to give better results than the old method of wiring.

Constrictive pericarditis has long been treated successfully by the surgical release of the heart.

Recent work done in the surgical relief of arteriosclerotic heart disease gives promise that a great number of these patients may eventually be given many more years of useful life. The present method of attack is by anastomosis of the coronary sinus to the aorta supplying the cardiac muscle from the venous side of the circulation.

Mitral stenosis is now being relieved surgically in a few patients with fairly good results.

No longer is it necessary for the child with congenital heart disease to suffer invalidism for years and then die. Cardiac catheterization may be used to analyze the oxygen content of the blood in the right side of the heart and to determine pressures within the heart. Such a procedure combined with angiocardiography allows accurate diagnosis in congenital heart disease. The tetralogy of Fallot is relieved by shunting arterial blood through the pulmonary circuit either by the anastomosis of a subclavian artery to the pulmonary artery or the establishment of a patent ductus arteriosus. The patient with a patent ductus arteriosus can be relieved by its closure. Coarctation of the aorta can be relieved by resection of the constricted portion. In the near future it may be possible to close septal defects in the heart.

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TEXAS SECOND IN POLIOMYELITIS FACILITIES

A nationwide survey of 6,276 hospitals registered by the American Medical Association shows that Texas ranks second to New York in the number of hospitals admitting poliomyelitis patients for treatment.

The survey, completed by the A.M.A.'s Council on Medical Education and reported in the November 19 issue of *The Journal of the American Medical Association*, reveals that 146 of the 1,243 hospitals in the United States which accept poliomyelitis patients for treatment are in New York and that 76 are in Texas.

PULMONARY NEOPLASMS, A SYMPOSIUM

I. Considerations in Their Earlier Apprehension

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THE problem of primary carcinoma of the lung is that now encountered in all efforts for cancer control—earlier diagnosis. A more universal appreciation on the part of layman and doctor of the magnitude of this problem, together with a knowledge of the encouraging developments leading toward its solution, will create interest and some measure of optimism.

Bronchogenic carcinoma is a common type of malignancy, being preceded in frequency only by cancer of the gastrointestinal tract, skin, and prostate. It is susceptible to cure in a significant proportion of cases if the patient is seen while the growth is still confined to the lung.

DIAGNOSTIC PROCEDURE

Although the roentgenogram of the thorax is the most important procedure for apprehending a pulmonary lesion, a thorough investigation based on the clinical symptoms may reveal the presence of a bronchial neoplasm in spite of a negative roentgenogram. The symptoms of pulmonary cancer, as well as the roentgenographic findings, depend upon the location, growth characteristics, and degree of bronchial encroachment. The appearance of a cough, or change in a chronic cough, may be an early symptom indicative of bronchial irritation. A localized wheeze suggests an incomplete bronchial occlusion. Recurring episodes of cough, fever, and thoracic pain and the expectoration of purulent and sometimes bloody sputum characterize a more advanced bronchial occlusion regardless of the cause of the obstruction. Weight loss, persistent pain, pleural effusion, and other manifestations found in detailed accounts of pulmonary malignancy are of late occurrence.

The radiographic manifestations of bronchogenic carcinoma vary from mass-like lesions and ill-defined areas of increased density through all the changes secondary to bronchial occlusion. The latter manifestations vary from that of obstructive emphysema and small segmental areas of atelectasis to those characteristic of pulmonary suppurative disease distal to an area of obstruction of a bronchus. Cavitation within a carcinomatous mass is not infrequently seen.

It is discouraging to note the delay in obtaining a roentgenogram after the onset of pulmonary symptoms. Overholt,[6] studying the time relationship between important steps which led to a verified diagnosis in 125 cases of bronchogenic carcinoma, reported that the average patient had seen a doctor within three months of the onset of symptoms but did not have a radiographic study for another three months. He reported further that the true diagnosis was not established until nine months after the first doctor saw the patient in spite of the fact that in 98 per cent of these cases, abnormal densities were demonstrated in the first roentgenogram. There can be no doubt that the temporary alleviation of symptoms by the universal use of penicillin has accounted for much of this delay.

Radiologic screening of the population now employed in the detection of tuberculosis in certain communities repeated at yearly intervals and extended to include the older age group would approach the ideal. Obviously it will be a considerable period of time before this method becomes universal. However, inclusion of a roentgenogram of the thorax as part of the examination of all patients having pulmonary symptoms, as well as those presenting themselves for a general medical examination, is within the realm of practicability. Recognizing that the greatest incidence (80 to 85 per cent) of pulmonary cancer is in men between the ages of 40 and 70,[1] the medical adviser can suggest that persons in this category have a roentgenogram of the thorax as part of a routine checkup. The reward would be a greater salvage of patients having this serious disorder.

It should be strongly emphasized that although the correct diagnosis may be suggested by the presence of an abnormal density on the roentgenogram, an exact diagnosis cannot be made from this study alone. Failure to appreciate this fact accounts for one of the great pitfalls in the establishment of an earlier diagnosis. A "wait and see" policy has given time for many pulmonary cancers to extend beyond the limit of effectual therapy.

When the appearance of trouble is noted on the roentgenogram, it remains for the institution of other procedures which are now well established for the determination of the exact diagnosis. Too often patients having bronchogenic carcinoma are given an erroneous diagnosis by means of radiographic findings alone. These erroneous diagnoses have included virus pneumonia, unresolved pneumonia, fungus disease, pneumonitis, and other conditions. The special

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diagnostic procedures available include bacteriologic study, special radiographic procedures including tomographic techniques, bronchographic studies, bronchoscopy, cytologic study, and exploratory thoracotomy.

Recently the examination for malignant cells of sputum, pleural fluid, and bronchial secretions and washings has proved to be one of the most valuable aids in the diagnosis of bronchogenic carcinoma, particularly for those lesions not accessible for bronchoscopic examination and biopsy.

EXPLORATORY THORACOTOMY

After a careful history, physical examination, and the employment of the aforementioned special procedures as indicated, there remains a significant number of cases in which the diagnosis is still in doubt and the condition must be classed as indeterminate pulmonary lesions. In lesions which are sufficiently localized, an exploratory thoracotomy may establish the diagnosis and provide for satisfactory treatment.

Recent articles have stressed the importance of this procedure.[2, 3] Johnson, Clagett, and Good[3] reviewed the records of 384 patients operated on for lesions involving primarily the lungs. On the basis of the clinical records, laboratory observations, and other diagnostic aids short of exploratory thoracotomy, a diagnosis could be made in 270 of these patients. In the remaining 114, or approximately 30 per cent of the 384 patients operated upon, it was necessary to resort to exploratory thoracotomy before a definite diagnosis could be made. The diagnosis in 3 of the cases remained indeterminate, even after exploratory thoracotomy, leaving 111 cases for study. These cases were observed over a period during which cytologic studies were not extensively employed.

These authors, chiefly on the basis of bronchoscopic and roentgenographic findings, divided the 111 indeterminate lesions into three groups. Group 1 included cases in which the predominant roentgenologic finding was a mass lesion in the lung, usually in a portion of the lung peripheral to the field of bronchoscopic visualization. Thirty-nine of the 53 mass lesions were proved at operation to be malignant. Group 2 included those cases in which the lesion was represented primarily by changes secondary to bronchial obstruction. In 24 of the 48 cases in this group, an underlying malignant growth was found at operation. A third, or miscellaneous, group of 10 cases included those instances in which a mass lesion could not be visualized roentgenographically and in which a definite bronchial obstruction could not be demonstrated preoperatively. In 2 cases, the lesion proved to be carcinoma; in 3, bronchial adenoma; in 3, suppuration and abscess; in 1, lipoid pneumonitis; and in 1, a silicotic stricture of the bronchus. The patients classified in the miscellaneous group were operated upon as an exploratory measure on the basis that there was no evidence of clearing by radiographic study after a few weeks' period of observation.

At this stage in a case, when the physician is confronted with a lesion falling in the miscellaneous category, a short period of observation is justified as a diagnostic procedure. In the presence of an underlying bronchogenic carcinoma, a positive cytologic study would prove helpful in eliminating this period of observation.

In considering the character of the lesions found at exploratory thoracotomy which are not of malignant nature, it is seen that surgical removal provides the only satisfactory treatment for even these. In other words, exploratory thoracotomy is not employed just to rule out malignancy. Although this is an important consideration, a patient with a lesion obstructing a bronchus which is not remediable by conservative means and with secondary changes in the pulmonary parenchyma has an unrelenting, downhill course, regardless of the nature of the obstructing process. It must be admitted that there are no good tumors and that they are all capable of growth with resultant encroachment on vital structures if not removed. Tuberculomas are prone to break down with dissemination of the disease. Irreversible suppurative processes are characterized by repeated clinical exacerbations until the body economy is finally depleted. Viewed in this broader sense, the procedure of exploratory thoracotomy becomes more rational and applicable in a far greater number of cases as well as providing the possibility of uncovering many pulmonary cancers otherwise obscure.

RESECTABILITY AND SURVIVAL

Statistics bearing upon mortality, resectability, and survival after pulmonary resection for carcinoma have to be reevaluated year by year. Many reports date back to that period soon after the first successful pneumonectomy for bronchogenic cancer was performed by Graham in 1933. Most reports show, however, that the extension or localization of the lesion and the pathologic cell type are the most important factors in survival after resection. Although improvements are still being made, and will continue to be made, the organization responsible for the surgical operation, including the operating team with its anesthesiologist, facilities for adequate preoperative and postoperative care, and the blood bank, can be considered relatively complete and adequate at present. It has been shown conclusively that the operative mortality rises in direct ratio to the exten-

sion of the lesion and the presence of secondary pathologic changes in the pulmonary parenchyma.

Ochsner and others,[5] in reviewing the records of 548 patients with primary pulmonary malignancy observed over the period from 1934 through May, 1948, reported that 1 of 3 patients had a lesion which obviously could not be resected, 1 had a lesion found at exploration not to be resectable (or the patient refused resection), and 1 had a resectable lesion. The gross survival rate after resection was 20 per cent at the end of three years with little change up to five years thereafter. If a patient survives three years after resection for bronchogenic carcinoma, the chance for cure is great. The mortality for the 1947-1948 series (10 of 61 cases) was less than one-third of the mortality rate for the first five year period of the analysis (12 of 22 cases).[4]

Jones's report in 1947 was encouraging. It was based on the experience of the preceding four years and included a total of 52 pneumonectomies with but 2 hospital deaths. This was accomplished by performing thoracotomy in 33 per cent of cases diagnosed and by carrying out pneumonectomy in 20 per cent of the total cases.

Provided the secondary pulmonary parenchymal changes are not too extensive, the risk for exploratory thoracotomy, as well as for a more conservative resection for benign lesions discovered at thoracotomy, is minimal. Aside from the chance for cure following pulmonary resection in cancer of the lung, the provision for palliation is considerable.

CONCLUSION

Intrathoracic problems are common and are of concern to all members of the medical profession. Most of the articles of particular help in offering a rational approach to these problems are found in periodicals devoted to the special branches of medicine. Obviously, it is extremely difficult for physicians to keep abreast of developments in all fields of endeavor. This unfortunate state of affairs is believed to account for much of the delay in the earlier apprehension of pulmonary cancer.

Many more malignant and nonmalignant conditions in the thorax could be remedied by widening the application of radiographic screening and by developing the concept of the urgent need for instituting the necessary procedures in a continuous chain of diagnostic events until the exact cause of a pulmonary lesion which has failed to resolve promptly, both clinically and by radiographic check, is determined.

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PULMONARY NEOPLASMS, A SYMPOSIUM

II. Diagnostic Value of Cytology Studies on Sputum and Bronchial Aspirations

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THE incidence of primary bronchogenic carcinoma is estimated by the various investigators to be from 5 to 20 per cent of all carcinomas.[11] In 1940, 5,556 deaths from carcinoma of the lung and pleura were reported in the United States.[15] Graham stated that the incidence is comparable to that of carcinoma of the colon and rectum[11] and reported that statistics from the St. Louis City Hospital and from Charity Hospital, New Orleans, showed carcinoma of the lung to be more frequent there than carcinoma of the stomach.[10]

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DIAGNOSTIC PROCEDURES

Since the first one-stage pneumonectomy was successfully performed in 1933, the early diagnosis of primary bronchogenic carcinoma has become of greater importance, and with improvements in technique hospital mortality following thoracic surgery has rapidly decreased. Graham reported only 2 deaths in 39 operations in 1947.[10]

Ochsner stated that "The most important factor in the diagnosis of primary carcinoma of the lung is the consideration of its possible presence."[16] Even after this important requirement is fulfilled, the exact nature of pulmonary lesions cannot always be established using all of the existing diagnostic methods. Roentgen-ray evidence may be inconclusive because of the coexistence of carcinoma and tuberculosis, lung abscess, pneumonitis, and so forth, and because of the cavitation which may be present in an estimated 25 per cent of carcinomas which are located in the periphery of the lungs.[2]

As reported by Adams,[1] Hammond,[12] and Bradshaw[4] positive bronchoscopic biopsies can be obtained in from 45 to 85 per cent of primary pulmonary neoplasms. Craver[6] and Gledhill[8] reported favorably on the use of aspiration biopsies in suspected lung carcinoma with accuracies of 51.6 and 78.5 per cent respectively. However, Ochsner,[17] Bergmann,[3] and Gowar[9] condemned the procedure because of the possibilities of hemoptysis, infection, pneumothorax, or extension of the tumor along the path of the aspirating needle. Thus, it is evident that additional procedures are needed to facilitate the detection of a pulmonary neoplasm in an early operable stage.

The examination of sputums for particles of malignant tissue is not new, Beale in 1860 having reported masses of carcinoma cells in the sputum of a patient with advanced carcinoma of the pharynx.[21] After this, isolated cases were reported but the consensus was that malignant cells could be demonstrated in the sputum in only a few advanced cases which were readily diagnosed by other means and that therefore sputum examinations were of no value clinically. These early investigators either examined unstained sputum or used only simple stains or embedding techniques similar to those for tissue sectioning.

In 1935 Dudgeon and Wrigley reported a series of 56 cases of lung carcinoma in which malignant cells were identified in the sputum in 43 cases.[9] This was accomplished using the "wet film technique" described by Dudgeon and Patrick in 1927 for the study of normal and pathologic tissue. A fresh morning specimen was examined carefully and blood-streaked mucus or solid tissue particles were selected, smeared, and fixed before drying in Schaudinn's solution. Hematoxylin and eosin stains were used. Despite clinically significant observations, the procedure gained little favor until after the appearance in 1943 of Papanicolaou and Traut's monograph, "Diagnosis of Uterine Cancer by the Vaginal Smear,"[19] when there was a renewed interest in exfoliative cytology.

CYTOLOGIC TECHNIQUE

The technique used by the various investigators at present is essentially that described by Dudgeon in 1935. A mixture of equal parts of 95 per cent alcohol and ether may be substituted for the Schaudinn's fixative, and the staining method of Papanicolaou for the hematoxylin and eosin. The proper collection of the sputum specimen cannot be overemphasized as it is largely the determining factor in the accuracy of this diagnostic aid. The patient should be instructed to cleanse his mouth of food particles and cough from "down deep" in the chest. A few cubic centimeters of material obtained in this way is preferred to a sputum cup full of saliva and nasopharyngeal secretions. In the presence of secondary infections, more satisfactory specimens may be obtained after antibiotic therapy. Although fresh specimens are preferred, 10 per cent formalin or 95 per cent alcohol may be added to minimize cellular degeneration and bacterial overgrowth when the specimen cannot be examined immediately.

Bronchoscopic aspiration of the mucus in the involved bronchus provides a high concentration of cells from the immediate region of the suspected tumor. The involved bronchus may also be "washed" with physiologic saline solution and the residue from the centrifuged specimen smeared and stained by the usual technique or embedded in paraffin and sectioned like tissue biopsy specimens. At least five smears on each specimen are prepared and systematically examined using a microscope equipped with a mechanical stage. Woolner and McDonald recommended the examination of three separate sputum specimens in suspected cases.[22]

CHARACTERISTIC CELLS

The normal cells encountered in smears of sputums and bronchial aspirations are derived from the blood, the squamous epithelium of the mouth and pharynx, and the columnar epithelium of the trachea and bronchi. Erythrocytes, polymorphonuclear leukocytes, lymphocytes, and plasma cells, frequently encountered in varying numbers, are readily identified.

Histiocytes or macrophages are sometimes more difficult to recognize because of their variable morphology. Characteristically, histiocytes are medium sized cells having abundant foamy or lacy cytoplasm and a fairly homogeneous, eccentric, round, oval, or bean-shaped nucleus. One or two small but prominent nucleoli may be present. The cytoplasm may contain phagocytized foreign particles, cellular debris, fat, or hemosiderin. The nuclei are sometimes large and deeply stained because of a dense chromatin structure, but the presence of phagocytized particles within the cytoplasm is a clue to the identity of the cell. Although these cells are usually mononuclear and

occur singly or in clumps, occasionally multinucleated forms having from five to twenty nuclei are seen. The presence of histiocytes in smears is a fairly reliable indication that the material is from the bronchial tree and not from the nasopharynx.

The squamous epithelial cells from the mouth and pharynx are wafer-like, polygonal cells having a large amount of homogeneous acidophilic or basophilic cytoplasm and small, dense, round to oval, centrally placed nuclei. Occurring singly or in small groups, the columnar epithelial cells of the trachea and bronchi are elongated pyramidal cells having abundant clear cytoplasm and a dense, round to oval nucleus, which is generally located toward the base of the cell. Sometimes two or three nuclei are present but are uniform in size and staining. Not infrequently, under reduced light, cilia may be seen on the free borders of these cells. The pyramidal shape may not be present when the columnar cells occur in large, closely placed masses, but they may assume a rounded or oval configuration. The nuclear uniformity and clear cytoplasmic envelope facilitates their recognition. Cuboidal cells with or without cilia may be present and closely resemble the columnar cells. Cells of the intermediate type occur singly or in small clumps and are not uncommon. They are moderate in size, round to polygonal in shape, and sharply outlined, and have abundant cytoplasm and a uniformly stained, round to oval nucleus. Basal cells are less frequently present and have a larger nucleus with proportionately less cytoplasm.

The malignant cells of the tracheobronchial tree share certain characteristics and, in addition, may have features which make possible classification as to type of malignancy. In general, the malignant cells are large, polymorphous, and more hyperchromatic than normal cells. Variations in the size and shape of the deeply stained nuclei exist, with few exceptions. There is a loss of the normal nuclear-cytoplasmic ratio; that is, the nucleus appears unduly large for the amount of cytoplasm present. The cytoplasm may be represented only by an incomplete rim having indistinct borders, or the nuclei may appear completely free of cytoplasm. Multiple and bizarrely shaped nuclei are not uncommon. The nucleoli may be single, large, and prominent, or multiple.

In squamous carcinoma of the lung the keratinized cells show a variable amount of cytoplasm, which takes a bright orange color with the staining technique of Papanicolaou. In the lower grades of squamous carcinoma, elongated nuclei are seen and epithelial pearls are not infrequently found. In the highly anaplastic squamous carcinomas, keratinization is not present, and the cytoplasm takes a basophilic stain. Phagocytosis by anaplastic cells of polymorphonuclear leukocytes and other anaplastic cells is occasionally encountered in smear preparations from squamous carcinomas.

The anaplastic cells from adenocarcinomas of the lung may occur singly or in small groups and may present a pleomorphic morphology. Although the amount of cytoplasm present may show an absolute increase, relatively it appears decreased because of the large nuclei. Although usually large, single, and deeply stained, the nuclei may be small and multiple. The nucleoli vary in size and number and, if single, are generally large. Vacuolization of the cytoplasm is variable but frequently marked.

The malignant cells from oat cell carcinomas of the lung are an important exception to the general observation of an increase in size of anaplastic cells in smears. These small cells are only two or three times the size of lymphocytes and may easily be overlooked when they occur singly. In small groups, the nuclear variation in size and shape is evident, and although the nuclei may appear finely granular, they stain deeply. The nucleoli are usually well defined and conspicuous but may not be seen. Little or no cytoplasm is present and the differentiation of these small anaplastic cells from lymphocytes and macrophages is sometimes difficult. Atypical cells which cannot be classified should not be reported as anaplastic, but only those cells showing the general or special characteristics outlined should be considered malignant.

ACCURACY OF METHOD

In 1943, using the Dudgeon technique, Gowar[9] reported a series of 93 cases of suspected carcinoma of the lung which were studied with a total of 238 sputum examinations, or an average of 2.5 examinations per patient. Of the 56 confirmed lung carcinomas, malignant cells were reported in the sputum smears in 36 cases. Of these, 13 lesions were considered to be potentially resectable but 5 patients refused surgery; of the 8 patients who were operated on, pneumonectomies were possible in 4. Of the 20 cases with negative sputums, only 5 lesions were considered potentially resectable and 2 of these patients refused surgery. The remaining 3 cases were subjected to exploratory surgery but the lesions were found not to be resectable.

Wandall[21] in 1944 published a comprehensive description of the cells occurring in sputum smears in both malignant and nonmalignant pulmonary lesions. In 100 proved cases of primary bronchogenic carcinoma, malignant cells were reported in the sputum of 84, and no satisfactory sputum specimens could be obtained in the remaining 16 cases. Bronchoscopies were performed in 82 of the 100 cases but positive biopsies were obtained in only 40. Twelve of the

100 lesions were found to be resectable and 10 of these patients had malignant cells in the sputum. Of the 82 cases studied by a combination of bronchoscopy and sputum examination, the diagnosis was established in 94 per cent. False positive reports were made in only 3.1 per cent of the 193 patients studied, 3 of these being chronic pneumonia and 3 tuberculosis.

Papanicolaou[18] in 1946 reported 52 cases having sputum smears. Of 31 proved carcinomas, 28 had positive or "suspicious" smears and 3 had false negative smears. The remaining 21 cases were diagnosed as negative both clinically and by cytology.

Herbut and Clerf,[13, 14] in 1946 studying smears of bronchial secretions only, reported 57 proved cases of pulmonary carcinoma, 47 cases of which were diagnosed by cytology.

Fremont-Smith[7] reported that McKay found anaplastic cells in the bronchial secretions of 40 of 54 cases of proved bronchogenic carcinoma. In 14 cases of this series, the bronchoscopic examinations were negative but the secretions were positive.

In an effort to stimulate the widespread use of sputum examination, Shatz, Bergmann, and Gray[20] investigated 40 consecutive cases of proved carcinoma of the lung and found that 29 cases showed malignant cells in the sputum. Five slides were made on each of 3 separate specimens, and the diagnosis of carcinoma was not reported on the basis of only a few isolated atypical cells but was reserved for those smears containing groups of obviously anaplastic cells. In the 29 cases having positive sputum smears, 7 had completely negative bronchoscopic examinations.

Woolner and McDonald[22, 23, 24] reporting on 2,188 patients stated that equally good results can be obtained with sputums or bronchial secretions. In this series, 200 positive smears were reported. Four of these were false positives; 4 were from carcinomas of the larynx, trachea, or esophagus; in 2 the final diagnosis was doubtful; and the remaining 190 were lung carcinomas confirmed histologically or clinically. Seventy-four cases were subjected to exploratory operation and in 29 the positive smears were the only preoperative microscopic evidence of carcinoma. In the 13 carcinomas removed, all had positive smears but in none of them could positive biopsy results be obtained. In 4 of the 13 removable lesions the bronchoscopic examinations were reported to be positive on the basis of fixation, infiltration, and appearance of the tumor mass, and in the remaining 9 cases endoscopic examinations were completely negative.

In April, 1949, Clerf and Herbut[5] brought their total of known bronchogenic carcinomas to 180, of which 161 or 89.7 per cent had positive smears from bronchial secretions. Biopsies were possible in 71 or 39.4 per cent, and in 42 or 23.3 per cent the bronchoscopies were reported positive on the basis of gross anatomicopathologic changes, making a total of 62.8 per cent having positive bronchoscopic examinations, as compared with the 89.7 per cent having positive cytologic smear preparations.

Although my own series of cases is too small to be of significance statistically, in the past fourteen months, of the 10 patients having positive smear preparations confirmed by tissue sections, 3 were operable and 1 is pending operation. There have been no known false positives. Two cases of false negatives are recorded, neither of which was confirmed by tissue study. In the first, a single sputum specimen was reported negative and the patient later died with a clinical diagnosis of primary carcinoma of the lung. Three negative sputums were reported on the second case having a clinical diagnosis of secondary carcinoma of the lung from a basal cell carcinoma of the face.

SUMMARY AND CONCLUSIONS

The literature on the cytologic examination of sputum and bronchial secretions is reviewed in an attempt to evaluate the clinical significance of this procedure in the detection of carcinoma of the lung. It is shown that a percentile accuracy of about 75 to 85 may be expected, and that a significant number of resectable cases may be diagnosed in this manner before gross changes detectable by bronchoscopy develop. It is suggested that a more widespread use of cytologic studies combined with bronchoscopic examination in suspicious pulmonary lesions will result in earlier diagnosis and thereby increase the percentage of cures in this important group of malignant tumors.

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PULMONARY NEOPLASMS, A SYMPOSIUM

III. Analysis of a Series Occurring on a Thoracic Surgery Service

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THIS study is based on a review of material from 130 cases of carcinoma of the lung. In many cases the tissue received consisted of a fragment removed bronchoscopically for biopsy. In other cases a lymph node or pleural nodule removed during exploratory surgery in which the tumor was found not to be resectable constituted the material available, and in many cases a lobe or entire lung was received. Material from 9 autopsies was studied and will be referred to although it was not included in the tabulation.

As noted by every observer, there was a marked predominance of carcinoma in men. Only 17 of our cases were women. Of the 17 women, 10 had adenocarcinoma. Of 110 patients with carcinoma (excluding adenoma) in which the age was recorded, the largest number, 36, were in the sixth decade of life; 35 in the fifth decade, 25 in the seventh decade, 9 in the fourth decade, and 5 were more than 70 years of age.

These tumors were classified as squamous cell carcinoma, adenocarcinoma, and undifferentiated carcinoma. The proportions of the various types was about the same as reported in other series.[7, 8, 11, 14, 19] Seventy-six tumors or about 55 per cent were classified as squamous cell type, 27 or about 25 per cent as undifferentiated carcinoma, 18 or about 10 per cent as adenocarcinoma, and 8 as bronchial adenoma. We did not encounter an instance of the rare supporting tissue tumors of bronchus or lung.

As numerous other observers have been, we were impressed by the varied structure of bronchogenic tumors, with regard not only to extension but also to the growth pattern in the gross and the histologic structure or figure.[3, 4, 20] In those cases in which the entire tumor was available for study, the occurrence of two distinct cell types was not uncommon and probably additional sections would have shown other instances.

SQUAMOUS CELL CARCINOMAS

Few of the tumors showed typical epidermoid squamous cell structure with distinct epithelial "pearls." The majority of squamous cell tumors were described as "nonkeratinizing." The better differentiated nonkeratinizing carcinomas reproduced recognizable epithelium, often with a distinct basal columnar layer of cells. The poorly differentiated carcinomas possibly might have been called transitional cell carcinoma, pleomorphic cell carcinoma, or carcinoma simplex by other pathologists. Brines and Kenning[4] have suggested that grouping these tumors with the undifferentiated or oat cell carcinoma is practical as regards rapidity of growth, liability to metastasis, and radiosensitivity.

Two tumors which we recently examined presented an interesting pattern. Both formed bulky, well demarcated, peripheral masses immediately beneath the pleura although not involving it. The structure of 1 was that of a highly pleomorphic anaplastic tumor having numerous multinucleated cytoplasmic masses. The histogenesis of this tumor was obscure but many areas suggested high grade squamous cell carcinoma. The structure of the other was similar but was more suggestive of squamous cell carcinoma and less pleomorphic. In neither case was another possible

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primary tumor found clinically, although in 1 case cerebral symptoms later occurred and identical tumor tissue was removed from the brain by a neurosurgeon.

ADENOCARCINOMAS

We classified as adenocarcinoma those tumors which showed in some areas definite gland-like complexes which might or might not be mucus secreting. The lining of the gland-like structures was columnar or highly irregular. We did not often find these adenocarcinomas to be peripheral in location, most of them originating near the hilum from a large bronchus. Many had already spread so extensively that the site of origin could not be determined. As has been noted in many series, approximately one-half of our cases of adenocarcinoma occurred in women in contradistinction to bronchiogenic carcinoma in general. Like other observers, we noted a tendency for this type to spread along the submucosa of bronchi, leaving the epithelial lining intact.

UNDIFFERENTIATED CARCINOMAS

Our tumors classified as undifferentiated carcinoma included a fairly distinct cellular type composed of small, round, or more often spindle shaped, cells. These were arranged in structureless sheets and rounded masses, separated by fibrous septums of varying thicknesses. Usually the spindle shaped cells showed pseudo-alveolar groupings. These cells, both round and elongated, had darkly staining nuclei and a narrow rim of cytoplasm. They displayed little variation in size, shape, and staining reaction and few mitoses. We did not include polymorphous tumors having larger irregular cells in this group as some authors have done.[7] Although we did not attempt follow-up studies on these patients, reports from elsewhere[9] and a clinical study by Lester, Shaw, and others[17] which included our cases indicated a tendency of these tumors to early and extensive metastasis, both lymphatic and blood borne. Our autopsy material, while scanty, justified confirmation of this view. We have seen tumor emboli in almost every organ, some of them only microscopic with no gross evidence of involvement.

GROSS PATHOLOGY

We were not impressed by differences in gross appearance of various cell types, other than those mentioned.

The majority of bronchiogenic carcinomas originate near the hilum in a large bronchus. If small, their bronchial origin is readily apparent; if bulky, a bronchus is merely seen to be involved in the mass. The smaller tumors are usually firm and fibrotic; larger masses almost invariably show wide zones of necrosis and hemorrhage. Some may show central cavitation. Adenocarcinomas have a tendency to grow as multiple masses in a lung or lobe. Bronchiogenic carcinoma in general shows a wide variation in manner of growth. Some tumors early form polypoid masses which grow into the lumen of a bronchus. Most, as they grow larger, form a roughly rounded mass in which the bronchial wall is eccentrically placed. Of great importance is the fact that hilar tumors, including bronchial adenomas, produce bronchial obstruction and a chain of subsequent events such as atelectasis, emphysema, chronic pneumonitis, bronchiectasis, and multiple or single abscesses in the segment of lung supplied by the bronchus. These secondary lesions produce symptoms and visible roentgen-ray changes, which more frequently result in diagnosis and surgical removal of the lung.

Probably the point most worthy of emphasis is the extremely obscure nature of a few of these tumors which early grow diffusely in the mucosa of a bronchus without forming localized masses or noticeable ulceration of the mucosa. These produce only a thickening of the mucosa and partial obstruction which is sufficient to cause marked peripheral changes of the nature of pneumonitis or abscesses. We believe that such diffusely growing carcinomas may easily be missed at autopsy unless carefully searched for in bronchi leading to chronically inflamed or atelectatic lungs, lobes, or segments of lungs. Many observers have mentioned the obscure nature of such tumors, but we believe the point deserves special emphasis.

In all except extremely small carcinomas, tumor emboli or thrombi in peribronchial lymphatics outside of the cartilages may be demonstrated. Somewhat further advanced cases show gross peribronchial extension evidenced grossly by a narrow zone of firm white tissue outside of the bronchus on sectioning. Later the tumor invades the lung parenchyma and while appearing grossly well demarcated, sections of the margin show lack of encapsulation or even of much reaction of the lung.

Tracheobronchial lymph node metastasis is, of course, frequently observed. The most bulky mediastinal masses we observed were in cases of undifferentiated cell carcinoma at autopsy. In some cases the tumor was directly continuous from the lung to the mediastinum. Such observations make understandable the fact that this tumor was formerly called "oat celled sarcoma" of the mediastinum.[2] Evidence of retrograde lymphatic embolism and metastasis, such as tumor emboli in subpleural lymphatics, has been observed only in extensive tumors, rarely in surgical specimens. We have, however, received surgically removed lungs with attached ribs or portions of chest wall which showed extensive pleural involvement.

ALVEOLAR CELL TUMORS

In recent years considerable interest has been focused on the rare tumor or tumor pattern of some tumors of the lung called variously "alveolar cell carcinoma," "alveolar cell tumor," "pulmonary adenomatosis," and lobar carcinoma."[9, 15] Much of this interest is probably due to the fact that a similar process in sheep known as jagziekte is apparently of virus origin and transmissible.[6] The histologic appearance of the human disease is striking in that the framework of the aveoli is left intact but is lined by a cuboidal to columnar layer of cells which may be stratified in some areas and in papillary folds in others. This arrangement may be observed in primary tumors of the lung, which uniformly show the characteristic makeup, in some areas of typical epidermoid or adenocarcinoma, and in tumor metastasis in the lung.[10] The origin of the tumor is unknown and theories concerning its origin are controversial.[10, 16] The theory that the tumor cells arise from alveolar lining cells is responsible for the term "alveolar cell carcinoma" and "alveolar cell tumor."[16]

We observed areas of "alveolar cell arrangement" in two tumors removed surgically. One was in an extensive tumor of the upper lobe of the left lung, involving the chest wall. Most sections of this tumor showed a fairly typical adenocarcinoma of the usual type. The other was a small peripheral tumor which in many areas was typical of bronchial adenoma. This tumor was followed roentgenologically for eighteen months and increased little or not at all in size. Because of the rarity of the reported association of alveolar cell pattern with bronchial adenoma, this case, with another observed at autopsy, is being reported separately by Cheek and Muirhead.[5]

We observed many areas having an "alveolar cell" pattern in material from several autopsies, both in primary lung tumor and in small metastases, but we have not studied a primary lung tumor in which areas typical of the usual cell types could not be demonstrated. We have studied metastases from 2 primary lung tumors demonstrating wide areas of alveolar cell arrangement; one of these having zones of mixed epidermoid and adenocarcinoma appearance, the other having zones typical of the "carcinoid" type of bronchial adenoma. In both cases metastasis in lymph nodes, liver, suprarenal glands, and kidney exhibited an alveolar cell arrangement with delicate fibrous septums lined by columnar epithelial-like cells. As several authors have suggested, bronchiogenic or metastatic carcinomas may merely utilize the existing framework of the lung as a scaffolding along which to grow.[13] Why tumor metastases from primary carcinoma of the lung should produce the same pattern in liver, suprarenal gland, or lymph nodes is less readily explainable. Of our 2 tumors with widespread "alveolar cell" growth, 1 gave the "multiple nodular" type of lung involvement; the other, a diffuse or pneumonic type of involvement,[7] although a circumscribed mass involving a large bronchus in the hilus was noted.

BRONCHIAL ADENOMAS

Bronchial adenomas constitute a small group, estimated at from 8 to 10 per cent of all primary bronchopulmonary neoplasms. Our series included 8 cases, a slightly smaller percentage than would be expected. Recognition of this lesion, however, is important because of the young age group usually affected and because of the good prognosis after surgical treatment, in contradistinction to the poor prognosis for bronchiogenic carcinoma.

Although this tumor is well recognized as an entity it has been called by many names: polypoid adenoma, basal cell carcinoma, adenomatous polyp, benign glandular bronchogenic tumor, carcinoid, mixed tumor, anlage, and rest tumor. Although the name "bronchial adenoma" is open to objections, we will refer to the tumor by that name because of its wide usage. This tumor may metastasize, but its usual slow course is well known so that it is always considered separately in reporting results of treatment of bronchogenic neoplasm. The origin is undetermined and various ideas have been advanced including origin from fetal lung buds, special cells called oncocytes,[18] bronchial ducts, and bronchial glands. Our observations produced no evidence for any one theory.

The incidence of this tumor in young persons and its relative frequency in women have been noted many times. Our series included 4 men, 1 of whom was a Negro; and 4 women. Two patients were more than 50 (58 and 63), 1 was 42, and the remainder were younger than 36, 1 being 20. The gross appearance of this tumor has also been frequently described, the tendency to involve large bronchi to produce an "iceberg"-like mass mostly peribronchial and the tendency to leave an intact mucosa over the mass being frequently noted. Probably worth emphasizing is the fact that the growth produces bronchial obstruction; the symptoms are therefore those of bronchial obstruction, the same as the usual symptoms of bronchiogenic carcinoma. Both may ulcerate and cause hemoptysis. One of our cases was peripheral and failed to produce the usual symptoms, being found on routine roentgen-ray examination of the chest. A long history frequently leads the clinician to suspect the diagnosis before operation even though the tumor cannot be seen through the bronchoscope.

We found, as noted by Holley[12] and others, that these tumors are readily classifiable into one of the

two general cell patterns, the "carcinoid" and the "mixed tumor." The carcinoid type resembles carcinoid tumors of the intestinal tract; in some the similarity is striking. The mixed tumor type resembles the epithelial component of mixed tumors of the salivary glands. Some of the latter type have been called mixed tumors of bronchus. We saw areas of both in 3 of our cases. The connective tissue stroma is frequently abundant in the mixed tumor type and in our material showed some areas of apparent myxomatous type and some areas of osteoid material. We did not see metaplastic bone formation in these tumors or observe cartilage other than the bronchial cartilages. These tumors show frequently but not constantly vascularity and rarely necrosis. The cells of the carcinoid type frequently show a pseudo-alveolar arrangement and may closely simulate glandular tumors. Of help in the differential diagnosis of adenoma is the retention of polarity, the lack of anaplasia, and lack of mitotic figures.

Our experience emphasized the difficulty of correct tissue diagnosis of bronchial adenomas as several of our cases were originally classified otherwise, one extremely vascular adenoma as a malignant vascular tumor. In 1 case, the long survival of a patient (seven years) after pneumonectomy prompted the surgeon to question the original diagnosis of undifferentiated carcinoma and ask that the slides be reviewed. On review the tumor was classified as a bronchial adenoma. The difficulties in diagnosing a small bronchoscopic biopsy specimen, which is usually ulcerated and infected and frequently hemorrhagic, are obvious.

SUMMARY

Tissues from 130 cases of primary lung tumors, removed surgically, are reviewed. The pleomorphic nature and occasional occurrence of two or more distinct cell types in the same tumor are reemphasized. The high incidence in men and in persons past 40 years of age is noted. The tumors are classified according to a widely used system into epidermoid squamous cell type, adenocarcinoma, undifferentiated carcinoma, and bronchial adenomas. Observations on growth, gross characteristics, and especially the appearance of extremely small, diffusely growing carcinomas are given. The peculiar tumor or tumor growth pattern known as alveolar cell carcinoma is described, and examples of this pattern observed in surgical specimens of lungs and autopsy material are cited. Observations on 8 cases of bronchial adenoma are summarized and difficulties of correct tissue diagnosis noted.

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ABSTRACT OF DISCUSSION

DR. LAUREN V. ACKERMAN, St. Louis: These three papers on bronchiogenic carcinoma have adequately shown the increased complexities of this problem. With increasing skill in radiologic technique and with increasing awareness on the part of the physician, interpretation of films in which the diagnosis of bronchiogenic carcinoma has arisen, has become increasingly more difficult.

In peripheral lesions, cytologic studies may help resolve the diagnosis; bronchoscopy is usually of little value. At the time of exploration, resection of the mass with frozen section seems to be a method of choice in diagnosis. After frozen section, the decision can be made as to whether pneumonectomy or local resection is indicated.

When the lesion begins in the main stem bronchus, the first sign as shown by Rigler is an area of localized emphysema which can be brought out by taking films at expiration and inspiration. In the expiration film, the area of obstructive emphysema will remain lighted up. At times tomograms will then resolve the diagnosis. With carefully trained personnel, the cytologic diagnosis of bronchiogenic carcinoma contributes greatly to the total diagnosis in a group of suspected bronchiogenic carcinomas. In the resectable bronchiogenic carcinomas, the number of cases which will have a positive bronchoscopic biopsy will usually not be more than 40 per cent. Cytologic studies of sputum and bronchial secretions will materially increase the number of positive diagnoses. If carcinoma cells are positively identified in the sputum or washing by the pathologist, the accuracy of this statement should be about 95 per cent. In a given group of cases of bronchiogenic carcinoma, the cytologic diagnosis will be positive in between 60 and 80 per

cent of instances. The increasing yield will depend on many factors including the stage of the disease, the capability and thoroughness of cytologic study, and most important of all, the method by which the sputums and bronchial washings are obtained.

The cytologic technique must remain as only one method in the diagnosis of bronchiogenic carcinoma and should be added to other findings in evaluating a diagnosis in a given case.

DR. JOHNSON, closing: Pneumonectomy is the operation of choice for bronchogenic carcinoma. There is a place, however, for a less radical procedure in certain cases, especially in those where there is limited vital capacity and where palliation is the chief end to be gained.

TUMOR SEMINAR

Neoplasms of the Lymphatic System

Conducted by

SHIELDS WARREN, M.D., Boston, Massachusetts*

DR. B. F. STOUT, San Antonio, Chairman: I have always found the Army to be willing and eager to help the civilians in any worthwhile projects. Today we have the honor of being invited to meet here at Brooke General Hospital and I am going to ask the commanding general of the hospital to address you.

Brig. Gen. Paul H. Streit: It gives me pleasure to greet the San Antonio Society of Pathologists at this, their fifth annual tumor seminar. Brooke General Hospital is greatly honored that you meet with us today. I realize that this is not a little local society; it is attracting attention in medical circles far and wide. I see visitors here this morning from New York, Memphis, and many out of the way places. I noticed articles regarding this meeting in *The Journal of the American Medical Association* and in several journals connected with the study of pathology. I am particularly delighted to greet Dr. Shields Warren, who is an old friend of ours and an occasional visitor at the hospital. I am sure you as well as I feel that he is a worthy successor to the previous guest conductors of this seminar.

We have an active residency training program at this hospital. It is only two or three years in age, but the staff and the visiting staff of the hospital are conducting it in an enthusiastic, intelligent, and splendid manner. Meetings such as this we are having today stimulate this program, add enthusiasm and knowledge, and help make the residents in training here study with more zeal and energy toward their special board qualifications. Again I bid you welcome and I hope you have a profitable and pleasant day.

Dr. Stout: It is now my pleasure to introduce to you the chief of laboratory service of Brooke General Hospital, Colonel Elbert DeCoursey.

Colonel DeCoursey: It is a pleasure to have you with us. I want to tell you that what Dr. Stout said about the help given by our laboratory reflects credit on the work of my staff, particularly Major Gilbert B. Stansell, the senior resident, who has spent not only day hours but many night hours getting ready some of the things that I hope you will like later. I also want to thank Lieutenant Gerald H. Lowe, my administrative assistant from the Medical Service Corps, who has arranged for supplies and equipment and setting up this room.

The man we are about to hear fills many places; you know him as one of the professors of pathology at Harvard University, pathologist at the New England Deaconess Hospital, and the director of the Division of Biology and Medicine of the Atomic Energy Commission. He never stays in one place. The fact is, I saw him last week in Washington, and while I was getting from Washington to San Antonio, he was going to Seattle, Minneapolis, and several other places on his duty. He is not only a man whom you can call a civilian, but we in the Army have him as a consultant to the Army Institute of Pathology. At the same time he was a consultant in the Army, he was on duty in the Navy; so, long before our lawmakers thought of merger, he was already merging at least one man into many military parts. I had the pleasure of being with him in Japan and at Bikini, where again we merged the Army and Navy without benefit of higher command.

It is indeed a pleasure to welcome Dr. Shields Warren. I think we can all be grateful that such a man is in charge of the medical section of the Atomic Energy Commission. How badly such a thing could

* *Assistant Professor of Pathology, Harvard University Medical School; Pathologist, New England Deaconess Hospital; Pathologist, Cancer Commission of Harvard University; Director, Division of Biology and Medicine, Atomic Energy Commission.*

EDITOR'S NOTE: *The tumor seminar conducted by Dr. Warren in San Antonio on October 23, 1948, was the fifth such annual conference sponsored by the San Antonio Society of Pathologists with the cooperation of the pathologists of Brooke General Hospital. The material for study was supplied by Dr. Warren and the San Antonio and Brooke General Hospital pathologists. Pathologists from throughout the state and elsewhere attended the seminar and were furnished histories of the cases under consideration. The microscopic slides were shown by lantern projector and each case was discussed by Dr. Warren.*

go you can imagine; to have a man we can trust and to know that the medical portion of atomic energy will go along the lines that we all want is really something. My personal opinion is that Dr. Warren knows more about the effects of radiation on human tissues than anybody else alive. Dr. Warren. (Applause)

Dr. Shields Warren: I am a little abashed by this introduction. I particularly welcome the occasion to be with you because it gives me an excuse to do some pathology again. I find that looking after chain-reacting piles and the migratory habits of salmon in the Columbia River and other things is pulling me away from the regular work in pathology.

I hope that in these slides, which I have found puzzling and stimulating, we will have opportunity for a considerable amount of discussion. If I am wrong, as I probably am in some of the diagnoses, I hope that you will be ready to set the diagnosis straight. For many of these slides I am sure more than one diagnosis has to be seriously considered, and the one that I happen to have set down may be far from right.

GRANULAR CELL MYOBLASTOMA

CASE 1.—Contributed by Dr. Warren.

Patient.—57 year old man.

Specimen.—From tongue.

The specimen consists of Zenker-fixed tissue measuring 2 by 1 by 0.7 cm., one surface of which is covered with fine papillary projections, in the center of which there is an 0.5 cm. center of ulceration with a firm, white base. The cut surface reveals that this lesion extends down to within 0.1 cm. of the receptive margin.

Dr. Warren: The first case presents an interesting type of diagnostic problem. This was a papillary lesion of the tongue in a 57 year old man. In the order of probability the pathologist puts the standard papillomas of the tongue first; then he considers the possibility of a malignant epithelial tumor in any papillary lesion, particularly when that papillary lesion is ulcerated and has a firm white base underlying it.

This particular slide was picked out, not because it was ideally representative of the lesion, but because it is so characteristic of the biopsy specimens that some of the surgeons often provide. The most readily accessible part of the lesion was obtained for study, and I think that the surgeon was perhaps more concerned with not having to put a suture in the tongue than with getting a really adequate sample of the growth. One thing that sometimes cannot be done tactfully but none the less is important is to be sure to let the surgeons know if we have not got adequate biopsy samples. I think that struggling too hard to diagnose a small biopsy specimen may result in more grief for the patient in the long run than getting a more generous and adequate sample of the tissue at the start.

The lantern slide shows one small portion of the papillary projections. I picked out these two papillae as being the most representative and most interesting portions of the slide. There are large cells which are distinctly granular in texture with one or more closely packed nuclei lying in the subcutaneous tissue and under the epithelium. Under the epithelium is a series of similar cells, large, granular, with single nuclei, and no mitotic activity (fig. 1).

Have we the scores on the diagnoses for this section?

Major Gilbert B. Stansell, Brooke General Hospital, Fort Sam Houston: The score in this case is granular cell myoblastoma 10 and lymphatic leukemia 1.

Dr. Warren: I side with the majority and put this specimen down as a granular cell myoblastoma. This was a distinctly unfair slide, in that so small a portion of the tumor showed. I picked it out simply to emphasize the difficulty that we as pathologists frequently have with the small biopsy specimens that are sent in.

Now as regards the question of lymphatic leukemia. There are lymphocytes infiltrating the submucous area. By and large I would say that submucosal infiltration in lymphatic leukemia is much rarer than is cutaneous. What is your feeling, Dr. Stout? How often have you seen infiltration of the mucous membrane?

Dr. B. F. Stout, San Antonio: I have never seen a leukemia of the mucous membranes.

Dr. Warren: Dr. Stout's experience agrees with mine. Why this variation should occur I do not know.

I think it is always dangerous ruling out cases on statistics alone because all of us have had the experience of the odd case that will turn up in spite of statistics. None the less, I think that it is worth bearing them in mind.

Secondly, in ruling out lymphatic leukemia, the scattered character of the lymphocytic infiltration is significant. Usually there is a fairly dense scattering and it is closely perivascular. Also, in this slide there is an abundant vascularization and there is no increase of the lymphocytes noted within the vascular walls in this area.

From the positive standpoint, it is well to remember that the granular cell myoblastoma occurs fairly commonly on the tongue and when it does it is almost invariably accompanied by this hyperplastic papillary reaction in the overlying epithelium. I do not recall more than one or two instances of myo-

blastoma in the tongue when this overgrowth of the papillary epithelium failed to accompany it.

This slide brings out the papillary overgrowth and the hyperplasia of the keratinizing elements. These compact masses of the light staining granular cells are about the only ones that show in this particular specimen. The subsequent excision of the lesion itself, the firm mass that underlay the ulcerated area, gave the typical picture, and when stained for the glycogen, gave an abundant amount of a glycogen in the slides.

I have never seen an adequate explanation for the high frequency of these lesions in the striated muscle of the tongue, but a highly disproportionate number of the reported cases have occurred in the tongue. Few of those occurring in the tongue have been malignant. Practically all have been benign even when they have ulcerated, as in this case, and have been fairly extensive.

Are there any questions or comments with regard to this particular case? How many of you have had granular myoblastomas from the region of the tongue in your own experience? (Show of hands.) That is a wide representation. It is interesting that the character of this lesion was not adequately recognized until just after World War I. Abrikossoff[1] first reported on the lesion in 1926. There was a good deal of interest in this type of lesion in the middle 1930's and the early 1940's and you will find fair scattering of reports in the journals during those years.

Dr. B. F. Stout, San Antonio: Do you regard this growth as a tumor or a degeneration of muscle?

Dr. Warren: I am glad you raised that point. I think we can cover at the same time the question of the rhabdomyoma of heart muscle because I think the processes are fairly comparable. I am inclined to think that this is a tumor of the hamartoma family rather than one of the ordinary run of neoplasms. In a fair number of these, where careful history has been obtained, an abnormality since birth has been noted. I believe that it represents essentially a malformation, a maldevelopment, only to a slight extent progressive, and that it falls into the true tumor group rather than the degenerative. If we contrast these cells with those of other degenerative changes in muscle, as for example in the muscle that has been deprived of its innervation through poliomyelitis, or the muscle that is undergoing various types of degenerative changes as in myasthenia gravis, or as in the various types of muscular dystrophies, we do not see this localized type of fairly specific change. The work of Wohlbach[2] a number of years ago on the rhabdomyomas of striated heart muscle was rather significant on this point. It gave evidence that at least certain of the granules are related to the nuclear complex, and from them the development of the characteristic striation lines of the striated fibers may subsequently come. All of you remember the beautiful spiderweb-like pattern that is seen in the rhabdomyomas of the cardiac muscle tumors. These striations are very rarely encountered in granular myoblastomas. In the malignant forms of the myoblastoma, all gradations up to a readily recognizable rhabdomyosarcoma may be encountered.

LYMPHOID TUMORS

I would like to show next the lantern slide that we had planned for the first lymphoid tumor. This is a schematic view that I thought might be helpful from the orientation standpoint, so that you would have an idea of the general terminology that I was shooting at in these lymphoid tumors. One of the difficulties that we all have in understanding the lymphoid tumors is that frequently we are not speaking exactly the same language with regard to them.

This slide shows a schematic drawing in which I have attempted to show at the top the most primitive type of lymphoid structure and at the bottom the better differentiated. In other words, this is sort of a synthetic lymph node which progresses from the least differentiated form at the top to the best differentiated form at the bottom. These actually are taken not from a developing lymph node but from different tumors of lymph nodes.

At the top we see the poorly differentiated syncytial or multinucleate, or even pseudoepithelial type of cell, with a large, rather vesicular nucleus, one or more condensations of chromatin, and light powdery chromatin in the rest that represents the primitive reticulum of the lymph node. The original structure of the lymph node is a cluster of syncytial cells of reticulo-endothelial origin, rather less differentiated than a true reticulo-endothelium, of endothelial origin, which, as they become better differentiated, will develop reticular fibers and will take on the form that we see a little lower down in the illustration. Sometimes vacuoles will form in the cells, sometimes not. They become more like epithelial cells in arrangement, the nucleoli a little more prominent, and they tend to line up in an almost pavement-like order. From them, in a less degree of differentiation than the mature lymphocytes but still partly differentiated, we begin to get a scattering of the mature lymphocytes. Then come the less mature lymphoid cells, of the type seen in the lymphosarcoma or in the acute leukemia of lymphoid origin, the lymphosarcoma with the scattering of the reticular cells still present. Next comes the more typical lymphosarcomatous picture of the poorly differentiated form of

lymphoblasts, and finally the adult lymphocyte of the malignant lymphocytoma at the bottom.

This is just a schematic representation, but it brings out one important point: These different tumors, although we name them by the predominant cell, are not pure—of one cell type. Just as we have an occasional adult lymphocyte in the early stages of differentiation of a node and just as we have some of the typical reticular cells persisting in the better differentiated form of the node, we will have in the tumors a scattering of different cell types. That is one reason there is so much confusion in the terminology of these lymphoid tumors. There are those who, if they see any reticular cells in a lymphoid tumor, will call it a reticulum cell tumor; there are those who, if they see any adult lymphocytes, will call it a lymphocytoma; and then there are all ranges between. Just as a convenient rule of thumb I have been using a classification that follows the embryologic development of the node. You remember that probably our most helpful aphorism in oncology is that the real basis of understanding tumors is understanding of the developmental phases of the individual cells. I think it was Rokitanski who first said that oncology is a recapitulation of ontogeny.

I speak of a tumor that has cells of predominantly this general characterization as being of the reticulum cell type. In the least differentiated form there are no reticulum fibers, and I have no hesitancy in making a diagnosis of reticulum cell sarcoma in some instances if there are these immature and syncytial cells, even though there is no formation of reticulum fibers. After the reticulum cell sarcoma comes this poorly differentiated form, the lymphosarcoma or, as it is sometimes spoken of, the malignant lymphoblastoma and then the best differentiated form, the malignant lymphocytoma.

These tumors have their parallelisms in the nonfixed tissue, the blood, in (1) the acute undifferentiated monocytic type of leukemia, (2) the acute lymphoid leukemia group, and (3) the chronic lymphatic leukemia group.

I am not including in this schematic presentation two other types of lymphoid tumors: (1) Hodgkin's disease, which I consider as a tumor and not as a granulomatous process and which I regard essentially as a tumor derived from the reticulo-endothelial cells; and (2) macrofollicular lymphoma, which I regard as a border-line lesion between the granuloma and the true tumor, which almost invariably winds up in a true lymphoid tumor.

One other thing that adds a great deal to the confusion of the diagnosis of lymphoid tumors is that these tumors will not stay put. I might cite as an example a man from whom I had a biopsy specimen in 1928, at which time the lesion was a typical macrofollicular lymphoma. At that time I was not sure whether it was a granulomatous lesion or a neoplastic lesion. I think now it probably was neoplastic. I did not recommend therapy for him and he had none. Subsequently, he did nicely, but he came in with a sharp enlargement of the nodes in 1936. We got another biopsy sample of the node which was characteristic for lymphosarcoma. No one would hesitate a moment on the diagnosis. He was given roentgen ray and responded reasonably well, with nice regression. He had clinical improvement and did well for three years. He had had several blood counts during this time and had no evidence of any abnormality, but he wound up in 1939 and died in early 1940 with a typical acute lymphatic leukemia.

Symmers pointed out when he first described this macrofollicular lymphoma the characteristic habit that it had of winding up in what he termed the polymorphous cell sarcoma, which sometimes looks much like one of the Hodgkin's pictures, which at times is a distinct lesion, sui generis, that does not fit into any of the ordinary types of lymphoid tumors, and which at times is a typical lymphosarcoma.

If we keep in mind this rough classification and the more or less labile character of these lesions, I think it will help us in considering this run of lymphoid tumors that we have to face.

One other point of importance: The average pathologist sees a fair number of lymphoid tumors but sees them on a selected basis. I think it is worth while for us to review briefly what the probable concentration of these tumors in the general population may be. The best figures in the case of leukemias that I have been able to find is that there are approximately 2 new cases per year per 100,000 living persons. This is including the childhood as well as the adult leukemias. I was anxious to get the right ratio on this because we are concerned with the survivors in Nagasaki and Hiroshima whom Colonel DeCoursey and I studied together and who are now being studied in prolonged and systematic fashion by the Atomic Casualty Commission.

In Hodgkin's disease, because of the long duration of some of the cases, it is difficult to get a comparable picture. Probably Hodgkin's disease is the commonest form of the lymphoid tumors, and my guess is that an estimate of about 4 to 6 cases per 100,000 persons per year would not be far from the truth. All of the other fixed lymphoid tumors lumped together would probably about equal the Hodgkin's cases.

Probably some of you have been interested in these statistical relationships. If so, I would welcome any comments that you might care to make. I know that a fair share of the clinical material of the Barnard

Hospital has fallen in the lymphoid group, and I wonder if Dr. Cooper would care to make any comments on what the prevalence of different types of lymphoid tumors has been in her material.

Dr. Zola K. Cooper, Oklahoma City: I do not have exact figures, but we would probably agree with your figures; we would get at least 1 or 2 new cases a year. As far as population is concerned, of course, our figures would be skewed a bit inasmuch as ours is a cancer hospital.

Dr. Warren: Yes.

In the Army we have one of the most valuable samplings of the young adult population that we have ever had, and the careful medical attention that the men receive is far above that of the general population. I wonder if Colonel DeCoursey would care to make any comments with regard to the Army's experience.

Col. Elbert DeCoursey, Brooke General Hospital, Fort Sam Houston: I am not familiar with the exact statistics concerning the findings. Probably the most tumors in the young men of the Army are in the lymphatic tumor group.

Dr. Warren: Any other comments on this general discussion of the lymphoid tumors? If not, let us head back to pathology again. This has been speculation more or less.

SARCOID

CASE 2.—Contributed by Dr. Warren.

Patient.—43 year old woman.

Specimen.—Spleen.

The patient was admitted in a semicomatose state. Physical examination revealed fixed pupils, impaired ocular movements above the horizontal plane, a choked right optic disk, stiff neck, left hypesthesia, diminished knee jerks, and no clonus or Babinski. On June 8, 1943, a ventriculogram revealed slightly dilated, symmetrical lateral ventricles, filled third and fourth ventricles, and no filling of the subarachnoid space. In the hospital, the patient ran a low-grade febrile course.

Dr. Warren: This case is one in which I expect a high batting average from the group. Major Stansell, how does it work out?

Major Gilbert B. Stansell, Brooke General Hospital, Fort Sam Houston: Boeck's sarcoid 8, Hodgkin's disease 1, lymphocytosis 1, and tuberculosis 1.

Dr. Warren: First rate! Let us see how the slides look.

These are good representative areas that have been picked out from this spleen. Sarcoid ordinarily occurs in lymph nodes. For that reason I picked out this sample of the spleen. This particular patient came in with splenomegaly as the chief complaint, and because there was no evidence of any leukemic change, because there was no evidence of any significant anemia, just the big spleen which was an inconvenience to the patient, the decision to do a splenectomy was reached. This was done uneventfully, and much to our surprise, in the section of the spleen, which was quite nodular, appeared these characteristic clustered lesions, which under low power are quite suggestive. In the higher power we see again the giant cells (fig. 2), and again under still higher power. And here the normal reticular net. This is the reticulum stain, showing that there is no particular fibrotic or reticular condensation about the lesion.

In this question of sarcoid, the differentials in the diagnoses that you have heard come up. First and most important, why is it not tuberculosis? Let us admit first that these are foreign body giant cells and not the Sternberg cells. When that happens we can automatically rule out Hodgkin's disease because while Reed-Sternberg cells will turn up in other things than Hodgkin's disease, I am always a little hesitant in making a diagnosis of Hodgkin's without finding the typical Reed-Sternberg cells.

Here are multinucleate cells of the foreign body giant cell type. In these cells there is a ring of nuclei all the way around the cell. That is rather common in the giant cell of sarcoid, and is rather unusual in the Langhans' cell which tends toward either having the horseshoe-shaped nucleus, or, if it is the aggregate in the center of an as yet noncaseating lesion, having the nuclei all the way through.

The second point that is sometimes helpful in these giant cells is the occurrence of the concretions. In Hodgkin's disease they are rare and they do not occur in tuberculosis. This particular slide, as I recall it, did not have the concretions within it.

The next point as to why this is not tuberculosis: Let us look at the granulomatous lesion. In this we see that there is no abnormal concentration of either collagen or reticulum about the lesion. In practically any tuberculous lesion except the youngest, there will be some concentration of collagen and reticular fibers.

Next, there is no evidence of any radially arranged reticular cells which are commonly seen about the young tubercle, or the old tubercle, for that matter. In the older lesions there is no caseation. In the younger lesions there are no polymorphonuclear leukocytes. In the older tuberculosis lesions, coagulation necrosis — caseation — is virtually the rule. In the younger lesions, practically always a few polymorphs are present even though actual caseation has not occurred.

One other type of lesions we need to differentiate from is the granulomatous lesion associated with some forms of the infection with Hansen's bacillus, leprosy. In this latter instance there are practically always the huge vacuoles filled with the organisms

that occur in the macrophages to help in the differentiation.

So here we have a lesion, granulomatous, noncaseating, no polymorphonuclear leukocytes, no peripheral fibrosis, no radial arrangement of the macrophages or reticular cells, with frequently several giant cells closely packed, with a ring of nuclei usually present about the giant cells. We are virtually forced to a diagnosis of sarcoid of Boeck's type.

Sarcoidosis restricted to the spleen is rare, but it is a lesion which turns up from time to time. In this instance we had a fairly marked enlargement of the spleen, which led to surgical removal of the organ. I think probably the patient would have gotten along just as well if the spleen had stayed in.

You notice from the history that in this particular case there was a distinct neurologic involvement. The neurologic involvement proved also to be due to sarcoid. This was a distinct widespread sarcoidosis of which we got an advance sample in this case of the spleen. It is rare that one finds as widely diffuse sarcoidosis as is encountered in a case such as this. Ordinarily it is restricted to a few clusters of lymph nodes. Autopsies of these cases are relatively few because the great majority of cases of Boeck's sarcoid mill along for a long time without getting into any appreciable grief. I have one case that I have been following for eighteen years without any appreciable change, without any appreciable grief from the first diagnosis that was made on axillary lymph nodes. The patient still has enlarged lymph nodes in his left axilla but he has not had anything beyond a gradual enlargement of them. Sometimes sarcoidosis involves the lung and it shows a roentgenologically diffuse, snowflake-like spattering throughout the lung. Whenever you find a granulomatous lesion in the lung with giant cells and with a roentgen-ray picture such as that, consider a diagnosis of sarcoid.

Beryllosis

There is one industrial disease which has only recently been recognized and which is important in this phase, and that is the lesion of chronic beryllosis. Beryllosis is a comparatively new lesion. How many of you have had cases in your experience? (No show of hands.) I am not surprised because there are relatively few. It is an occupational hazard, and the industry is fairly sharply localized. Beryllium is used primarily in three places. It is used by the fluorescent lamp industry to combine with other materials to give the coating of the fluorescent tubes. It is used as a beryllium copper alloy in industry to quite an extent, and it is used in certain phases of the operation of the Atomic Energy Commission. That is where I had to get interested in it. The first cases of chronic beryllosis, so far as I know, were recognized by Dr. Harriet Hardy, in Massachusetts.

Chronic beryllosis is an insidious disease which becomes apparent clinically by increasing lassitude and some shortness of breath. It is characterized by the occurrence in the lung of granulomatous lesions accompanied by fibrosis, which at first glance could be mistaken for sarcoid, except that particulate material can usually be found in the giant cells. Chemical analysis is necessary for making a final diagnosis. A history of exposure is helpful but is extremely unreliable at times. It is the most insidious disease with which I have ever had experience, and it is well to keep in mind our experience with beryllosis from the standpoint of industrial hazards in general.

In the first place, competent investigators of the United States Public Health Service tested beryllium in animals as recently as 1942 and 1943 and reported that it was harmless. It was, on the basis that they used it, but it was none the less a troublesome thing later to us. This report that it was harmless proved to be misleading. It was found that workers exposed to various phases of finely divided beryllium or beryllium oxide came down with an acute pneumonitis. In a few instances it was fatal; in other instances it was annoying but the patients apparently got well. Then it was recognized that some of the persons who had been exposed were coming down with these symptoms that I described: shortness of breath and lassitude, and on roentgen-ray study they were found to have an extraordinary picture that looked perhaps more like multiple minute metastases of a carcinoma than anything else. The picture was greatly confused by the fact that there began to be some off-site cases. There were patients living several blocks away from the plant who came down with the disease. In a recent survey that we have made jointly with the Ohio State Department of Health of one of the plants that supplies the Atomic Energy Commission, there was a discovery of 5 off-site cases, some being a quarter of a mile from the plant. One case, much to our surprise, picked up by routine roentgenograms of the general population was several miles away from the plant. We found on checking that this patient was the wife of a plant worker who washed his clothes, and undoubtedly he carried home enough dust to cause the disease. We have had 2 patients who were the wives of workers and washed the clothes in which the men worked.

Individual susceptibility plays an important part in any random group exposed. Only a relatively small percentage will develop the chronic form of the disease. Thus far it has been difficult to produce in the experimental animal, although there is some evidence that the experiments that Dr. Gardner at Saranac did shortly before his death and Dr. Vor-

wald is carrying forward at present may be approximating the chronic form in human beings. Curiously enough, pregnant women are exceedingly susceptible to the disease, and in the out-site cases a disproportionate number of cases occur in the pregnant woman. What the determining factor is there, we do not know.

We do know that if we maintain a concentration in and around the plants of under .0002 micrograms per cubic meter of air we are safe. One reason we know that is that when we picked Pittsburgh for a control area because of the tremendous amount of soot in the atmosphere, we found to our surprise that that was close to the concentration of beryllium in Pittsburgh air. Curiously enough, Pennsylvania soft coal has a slight contamination with beryllium. Thus we have in the absence of this disease in the general population in Pittsburgh good evidence that we have an actual safe standard to shoot for in air. These standards can be achieved in industry and we have accepted this as the standard for the time being in the plants having Atomic Energy Commission work to do.

I did not mean to get off the beam, but I thought a disease of this sort might be of interest. I think it is important as a pattern in indicating that we must be alert to industrial disease, not only from the standpoint of things that we know to be injurious, but also from things that we think are safe.

Dr. William W. Tribby, Memphis, Tenn.: In my slides I noticed some beautiful so-called asteroid bodies. I know that some people who have written on sarcoidosis consider that this is almost diagnostic of it. I would like to have your opinion.

Dr. Warren: You are more fortunate than some of the rest of us were. How many found asteroid bodies? (Show of about twelve hands.) Well, that is working out a lot better than I thought.

The question of the specificity of these asteroid bodies is hard to settle. When I see them, I think strongly of sarcoid. There are other things that can simulate them, particularly in the salivary gland areas. There are calcific concretions arising from the inspissation of salivary secretions locally that may be picked up by giant cells and have this asteroid type of arrangement. I am not aware of their occurrence elsewhere than in salivary gland areas or in lesions other than sarcoid. Dr. Stout, what has been your experience?

Dr. B. F. Stout, San Antonio: I have not seen them.

Dr. Warren: Dr. Tribby, what has your experience been?

Dr. William W. Tribby, Memphis, Tenn.: I have not found them in any other condition either.

Dr. Warren: They are not absolutely required for the diagnosis.

Dr. Leo Lowbeer, Tulsa, Okla.: I believe the diagnosis of sarcoid in this case is well established on account of the large number of foreign body type giant cells within noncaseating granuloma in the absence of fungi. However, if the pathologist sees granulomas with giant cells, less giant cells I would say, in the spleen, liver, and bone marrow, he should also seriously consider the presence of a brucellotic granuloma. The giant cells in brucellotic granulomas are extremely varied in structure. Some of them look exactly like those shown here with the nuclei all around the periphery of the cell. However, the giant cells are never as frequent in brucellosis as in this case.

Dr. Warren: That is a helpful point. I am glad that you spoke of it because I had overlooked the problem of the differential of brucellosis. Ordinarily the nuclei in the brucellotic granulomatous lesion will be much more irregular in outline than the fairly even well-rounded nuclei that are in the giant cells here.

Dr. A. O. Severance, San Antonio: In the slide I was looking at, in addition to these asteroid bodies I found in several of the giant cells little round structures with a clear area, in the center of them a little round dot, reminiscent of histoplasmosis. That is the reason I wrote that diagnosis down. I would like to know what you think they are.

Dr. Warren: In the small form as they are here, these are difficult to recognize. Actually they are probably small agglomerations of calcium carbonate. I think it is the refractility with the partial transparency of them that gives them this form that looks much like histoplasma; I agree with you on that. If you watch them you will find in other lesions—not in this particular case—a steady increase in size, and they will ultimately produce the round laminated bodies that are similar to those associated with some of the mucinous papillary cystadenomas of the ovary, concentric lines, looking like the contour lines on a map, of the calcific material that is laid down. I think that these small bodies you describe are the smallest recognizable form, the initial phase about which subsequent layers of calcification occur, leading to the larger calcific rounded masses.

Dr. John J. Andujar, Fort Worth: Was the tuberculin test positive in this case?

Dr. Warren: I am sorry, I cannot answer that point. At times the tuberculin reaction is confusing. I think one can get either a negative or a positive tuberculin reaction in these cases. I have also had

the disconcerting experience of finding actual tuberculosis present in relation to the sarcoid lesions as well. The evidence is fairly strong that there may be some relationship between tuberculosis and sarcoidosis, but I do not know of anyone who has figured this out. I have inoculated practically every animal from the frog, the snake, and the turtle up through to some of the primates in the hope of getting some sort of organism out of sarcoid, but I have never succeeded in it.

MACROFOLLICULAR LYMPHOMA

CASE 3.—Contributed by Dr. Warren.

Patient.—46 year old white man.

Specimen.—Lymph node, spleen, and bone marrow.

Three years before the patient noted lumps in his neck, axillae, and groins, but he did not seek aid until two years later. At that time he entered the hospital because of cough, dyspnea, swelling of the abdomen, edema of the legs, weight loss, and weakness. The white blood cell count was normal with a normal differential. Platelets were normal. The sternal marrow indicated erythropoietic anemia. Mediastinal lymph nodes were also enlarged. The patient responded to roentgen-ray therapy to the mediastinum and axillae, returning to work for one year before symptoms recurred and death ensued.

Dr. Warren: In this slide can be seen under low power an appreciable collection of small foci of rather large, somewhat similar cells.

Under the next higher power there are penetration of the capsule, some of the normal looking lymphoid cells, and scatterings of larger cells. Here is a higher power that brings out some of the less mature cells.

Let us see what our diagnoses were: Hodgkin's disease 3, myeloid leukemia 1, plasma cell leukemia 2, lymphosarcoma 4, plasma cell myeloma 1, and lymphatic leukemia 1.

A well distributed set of diagnoses and a case that it is well to consider carefully.

This case is one in which I think there is some reason for the varied types of diagnoses that we had. I know that in taking the photographs Major Stansell had to put a lot of attention on this case. Because it is always good to get a fresh viewpoint on these things and to get away from initial prejudices that I might have for my first diagnosis (it was my case originally), I wonder if we could get Major Stansell to discuss his impressions of the case.

Major Gilbert E. Stansell, Brooke General Hospital, Fort Sam Houston: I think that probably the original lesion was a giant follicular lymphoma involving the lymph nodes and the spleen, but the lesion in the bone marrow I am not too sure about. I feel that in the bone marrow with the myeloid hyperplasia and in the lymph node with the invasion of the capsule, I could not definitely rule out an unusual or peculiar type of early lymphosarcoma developing in this lymphoma. That was my impression, a giant follicular lymphoma with sarcomatous change, particularly in the lymph node, and myeloid hyperplasia of the bone marrow, which has not been shown yet. It is on the next slide.

Dr. Warren: Suppose we turn to the next slides now. This brings out the lesion of the spleen with again the large follicles and fusion of the follicles formed. One of the important points to my mind for the diagnosis of macrofollicular lymphoma is not only the large size of the follicles but the fusion of follicles, the evidence of breaking across the normal reticular barriers which exist between the separate follicles of the normal lymph node. In hyperplasia of lymph nodes no matter how big the follicles may get—and they may get pretty big sometimes—they remain discrete, crowding one another a little, but usually with a rim of normal lymph node stroma in between. Early in the course of macrofollicular lymphoma there will be a fusion of the follicles because of the invasion of the intervening stroma.

Here we see under somewhat higher power the general picture. This last picture is bone marrow. This also is bone marrow. You can see here the hyperplastic response. This is a good example of the puzzling type of response that sometimes occurs in these lesions.

My initial diagnosis was macrofollicular lymphoma, and on the first biopsy a year before the death and autopsy, we had a distinctive macrofollicular lymphoma that no one would question. Because we were much puzzled as to just how far the lesion had gone and what it was doing, we made slides of this and sent it around to a number of pathologists and to the Army Registry. We got back a variety of diagnoses, as one always does when one sends queer lymphoid tumors around. In general we came to the conclusion that this was the beginning of the Symmers' type of polymorphous cell change that occurs in the macrofollicular lymphomas and that there was in the marrow a myeloid hyperplasia.

Interestingly enough, a disproportionate number of our patients with Hodgkin's disease have later shown what apparently is myelogenous leukemia. I wonder whether this disturbance of the reticuloendothelial system in Hodgkin's disease may occasionally lead to a further disturbance that turns up as leukemia or whether the roentgen-ray therapy that the patients have received may have been provocative of leukemia. Physicians as a whole have five times as many cases of leukemia as the general population, and we are in the course of getting out statistics on the radiologists to see whether or not

they contribute chiefly to the physicians' high level. I have also been impressed that many more dentists, particularly those who take their own roentgenograms, are apt to turn up with leukemia. That is not on a statistical basis but just on chance observation. More than once I have gotten dentists to check their x-ray apparatus and have found that they are getting a good deal more x-radiation than they were anticipating.

Incidentally, have we any radiologists in the group? (One hand shown.) I would like to ask our lone radiologist what estimate he would give as to the dose of roentgen ray that would be received in taking a complete set of films of the oral cavity.

Lt. Col. Alfred Ackerman, Brooke General Hospital, Fort Sam Houston: The dentist would most likely receive quite a few roentgens because he deals with a soft type of radiation and with a high milliamperage output, but it is over a short time and over a small area, so that in spite of the roentgen measurement the actual dose, as far as the erythema effect is concerned, would be low.

Dr. Warren: That is a sound statement. We have made some measurements and find that the radiation will integrate at about 32 r. It is surprising how much scatter there may be at times, even though it is soft radiation. I think a good many dentists get considerably more roentgen ray in the scatter than they realize. They stand out of the way, but they do not make a point of standing as far away as they should. If I were doing that sort of work I would want to have my footswitch or my handswitch with a long enough lead so I would be at least 6 feet away from the patient. Certainly the kind-hearted practice some dentists used to follow of keeping their finger in to hold the film was bad not only from the standpoint of their fingers—I have seen some bad burns as a result of that—but also bad from the standpoint of their total body radiation as well.

Now to come back to this case: Do the advocates of leukemia here want to make a fight for what I will call a lost cause? Perhaps they are not willing to regard it as a lost cause.

Dr. B. F. Stout, San Antonio: Does the giant cell follicle type of lymphosarcoma often invade the bone marrow or is invasion of the bone marrow an indication that had the patient lived long enough the disease might have developed into a peripheral blood picture of leukemia?

Dr. Warren: The question as to whether the invasion of the marrow by the tumor is an indication of a probability of leukemia turning up is sound. I think whenever there is infiltration of marrow by any tumor—metastatic carcinoma, let us say—the chance of getting a leukemia-like reaction, a leukemoid reaction is a fair possibility. True leukemia under those instances I think is rare, and in this instance I would be inclined to think that we are dealing with a simple replacement of the marrow and a compensatory hyperplasia of the persisting hematopoietic tissue rather than a real preleukemic stage. Since a fair number of lymphoid tumors do wind up in leukemia, I may be off base in that statement; but from this, I would say that this particular man's chance of getting leukemia had he lived would probably not be greater than that of the average person.

HISTOPLASMOSIS

CASE 4.—Contributed by Dr. Warren.

Patient.—67 year old man.

Specimen.—Right ethmoidal sinus.

The patient had experienced discomfort from an upper denture for five years, but for one month before admission this had become acute, with swelling and tenderness of the right cheek and development of an ulcer-like cavity. Biopsies December 4, 1947, showed ulceration and chronic inflammation. Routine bacteriologic tests showed typical mixed mouth infection. Detailed local examination showed ulceration of the mucosa of the underlying bone and osteomyelitis of the maxilla. In February, 1948, the patient's right eyelid began to swell and the right side of his face was numb and swollen. He was admitted to the hospital with marked edema and extensive ulceration of the roof of the mouth which had extended into the nasopharynx.

Dr. Warren: I am expecting a wide variety of diagnoses here; frankly, I do not know myself what the right diagnosis is. This is a case that was misleading from the standpoint of the history. This 67 year old man had been having trouble with his teeth for a long time. Finally the situation became acute and at that time an ulcer became apparent in the inner aspect of the right cheek. Several biopsies showed ulceration and chronic inflammation. We wondered what type of organism might be involved because there is always the chance of some queer infection giving a picture of this sort. We found that there was evidence of destruction of the bone and of the maxilla, and that there was osteomyelitis of the maxilla. In spite of 4,200 r of roentgen-ray treatment given at a time when we had thought that this might be a neoplastic process, there was inadequate response. That dosage ought to hit the average tumor of lymphoid type pretty hard, but here we did not have any satisfactory response from it. We finally had the development of marked facial edema and extensive ulceration in the roof of the mouth, and the whole process had involved much of the right side of the nasopharynx as well.

Suppose we now have a look at the slides. Here is a close-packed mass of cells. You recognize the mucosal gland persisting and you notice the ciliated

respirated epithelium of the nasal mucosa persisting. This section came from the antrum, although it was obtained through the oral cavity.

Here you see the general histologic picture. Here is a reticulum stain with the irregular, rather profuse development of a network of reticulum fibers in and among these abundant cells. In any persisting granulomatous lesion there is always the problem of rhinoscleroma to consider. I understand that Dr. John Moore has had some cases of this lesion, and I wonder if he would describe briefly the findings in them and the significant points, as I think we want to consider that from the differential standpoint in this case.

Dr. John Moore, San Antonio: I have observed 1 case. There was a granulomatous lesion with the Mikulicz type of cells and also the hyalin-like bodies that are associated with this condition. The patient was treated with streptomycin and the lesion cleared up entirely in just a few weeks.

Dr. Warren: That is a satisfactory outcome. I remember seeing some cases a number of years ago in Switzerland that had originated in eastern Europe. There they had been chronic for some years and were slowly progressive. Of course until we had antibiotics they were pretty hopeless from the therapy standpoint.

Dr. M. Gerundo, Lubbock: I have had 2 cases of rhinoscleroma which were reported in the *Hawaii Medical Journal*. The principal cell I would say is the plasma cell. I think that the Mikulicz's cells show evidence on histologic section that they are mostly derived from the plasma cells, so that the fundamental cell, particularly in the early phase, is the plasma cell and not the Mikulicz's cell. Russell's bodies—or as they were called, the hyaline bodies—are degeneration or maturation of the plasma cells. Of course, it is possible that the plasma cell does contribute to the latter stage of sclerosis.

In this case we do not see any plasma cells, and I would be much disposed to say that it is not rhinoscleroma although the range of the same Mikulicz's cells can be found in leprosy. Occasionally in Hawaii I have effected a diagnosis which could be rhinoscleroma or it could be leprosy, so that it is definitely the bacteriologic stain which one should watch. As to therapy: In 1 case we gave roentgen ray and the lesion cleared without any antibiotics.

Dr. Warren: That is an interesting point. I had not realized that roentgen ray was helpful from the therapeutic standpoint. The diagnostic problem in this case, when it came through our laboratory, lay between lymphosarcoma and a granulomatous lesion. The advocates of lymphosarcoma gradually swung over to a reticulum cell sarcoma. They were influenced in large part by the character of these rather uniform cells (pointing to slides) with a fair degree of mitotic activity among them, and by the diffuse distribution of the new reticular net here. They were also influenced by the chronic course of the disease. They were a little worried by the fact that there was not adequate response to roentgen ray. I went through the same processes that they did when I looked at the slide. Then I was struck by some areas similar to this one up here where there are large foam-like cells with definite granules scattered in them.

In this case, which we will consider more carefully with relation to one of the later cases, I have come to a tentative diagnosis of histoplasmosis. I found a number of the large cells with organisms that resembled the histoplasma scattered through. I am inclined to think that it may be in the histoplasmosis group. On the other hand, it is difficult to say flat-footedly that this is not a lymphoid tumor.

Let us see what the score on this particular case was. Malignant plasmocytoma 1, Hodgkin's disease 1, reticulum cell sarcoma 4, undifferentiated carcinoma 1, hemangio-endothelioma 1, histoplasmosis 2, lymphoepithelioma 1. Well, I see that you are having the same difficulty that I have had with it.

In regard to the malignant plasmocytoma, there are few multinucleate cells here. By and large in the malignant plasmocytomas that I have seen from the oropharyngeal region there has been a strong clustering of cells with from three to six nuclei, a few of which of course can be found in normal plasma cells as well. In the absence of those and in the absence of the clear-cut histologic picture of the rather sharply defined well rounded ovoid cell, with the eccentric nucleus, with the halo of clear area to one side, I have been rather hesitant to make a diagnosis.

Hodgkin's disease I did not like to consider as a diagnosis here because of the fairly uniform cellular pattern on the one hand and the absence of Reed-Sternberg cells on the other.

Reticulum cell sarcoma I considered seriously, and I am not sure that I can even yet rule it out.

Undifferentiated carcinoma, I think the reticulum stain would rule out. I do not recall in any epithelial area having seen that pattern of reticulum. Reticulum is laid down around the clusters of epithelial cells primarily in a carcinoma rather than being scattered diffusely through them.

As for hemangio-endothelioma, this is a vascular lesion, to be sure, and there is active proliferation of young capillaries, but I do not think that we have the characteristic pattern of hemangio-endothelioma.

Histoplasmosis, as I say, I am considering seriously, and I think there are histoplasma there. I am not

quite sure whether it is the only thing in the lesion, or whether it is something else.

Lymphoepithelioma, the pattern of the reticular net again rules out.

Chronic inflammation I will lump with histoplasmosis and say we cannot rule out a chronic inflammatory basis. This is about as nearly borderline a lesion as any that we have in the group.

Are there any comments and questions with regard to this case?

Dr. Paul Brindley, Galveston: I would like for you to comment on the large number of mitoses in the nasal mucosal section, some of which were a little irregular in character.

Dr. Warren: I think we have to remember that this is a chronic inflammatory lesion; that there is a tremendous swelling and distortion of the contour of the nasal mucosa; and that there is undoubtedly a stimulus for rapid mitosis in order to attempt to keep an epithelial covering over this rapidly increasing mass. We also have the problem in regard to irregularity as to what radiation may have done. The effect of radiation on a lesion may show up in terms of irregularity of mitosis.

Dr. Cooper has had a great deal of experience in postradiation biopsies. I wonder if she would comment on the variation of mitoses that she sees under these conditions.

Dr. Zola K. Cooper, Oklahoma City: I am sure there is great variation. Multipolar spindles are sometimes found and a clumping of the chromatin of the chromosomes in many abnormalities in the spindle following radiation, and I think too, depending on the time after radiation, there may be an increase in mitoses immediately following it.

Dr. B. F. Stout, San Antonio: I looked upon this as a granulomatous lesion. In view of that fact I turned the oil immersion lens on the slide and was confident that I saw a small yeast that would be compatible with that of histoplasma.

Dr. Warren: I am glad to have that reinforcement.

Dr. Leo Lowbeer, Tulsa, Okla.: Has this lesion been cultured?

Dr. Warren: We unfortunately made cultures from the lesion in just swabs. We did not culture the actual excised lesion. We did not grind up a portion of the lesion and culture it as we ought to have done.

Dr. A. O. Severance, San Antonio: In support of the reticulum sarcoma school of thought here today, I would like to ask if we could not possibly have a primary neoplasm of a reticulum cell lymphosarcoma type, and then postulate that an ulcerated lesion invaded by a secondary yeast-like organism would give a picture that Dr. Stout has just described. I have seen 2 cases of histoplasmosis in the patient when the patient was alive, and the lesion simulated tuberculosis in both instances. A fungus was readily cultured; every time we tried we got a yeast-like organism which we erroneously thought was a Monilia albicans in culture and in the smears. The fact that you did not get anything to grow may be that it was not planted on Sabouraud's or some other media.

Dr. J. L. Goforth, Dallas: In the section that I studied on this case there were deep in the granulomatous tissue nests and small ribbons of cells that had the general appearance of epithelial cells. I wondered if they possibly represented a primary epithelial tumor that had become secondarily infected and had produced the granulomatous change. I further wondered whether these cells might possibly be enameloblasts.

Dr. Warren: That is a good point. I was inclined to interpret those nests of epithelial cells found fairly deep as a residual of the mucous secreting glands of the mucosa. The roentgenogram was not at all compatible with adamantinoma, and I was hesitant in interpreting cells as being of the group of the enameloblastic series unless there was the characteristic orientation of the nuclei with regard to the basement membrane. That is a constant feature of the average adamantinoma. I thought of an infected adamantinoma as one of the distinct possibilities here but that ruled it out.

Dr. D. A. Todd, San Antonio: The 1 patient with histoplasmosis whom I have seen—I believe Dr. Severance was in on this case—went up to the Mayo Clinic and there it was diagnosed as leishmaniasis.

Dr. Warren: That is interesting.

This case I think we will not try to pin a hard and fast diagnosis on, but leave it open as to whether the histoplasma are the primary factors here. I think probably—on morphologic evidence at any rate—we can just interpret these bodies as being compatible with histoplasma, and whether or not they are accompanied by a tumor. If it is a tumor, I think it is a tumor in the reticulum cell sarcoma group rather than in the other groups. The fact that there was no response with the amount of roentgen ray that the patient had I think is perhaps significant evidence in favor of the granulomatous origin, because on the whole it is rare to get a lesion fairly restricted that will not respond to 4,200 r of roentgen ray and still be a reticulum cell sarcoma. There are a few resistant ones but they are extremely rare.

Dr. B. F. Stout, San Antonio: Would radiation have any effect on histoplasmosis?

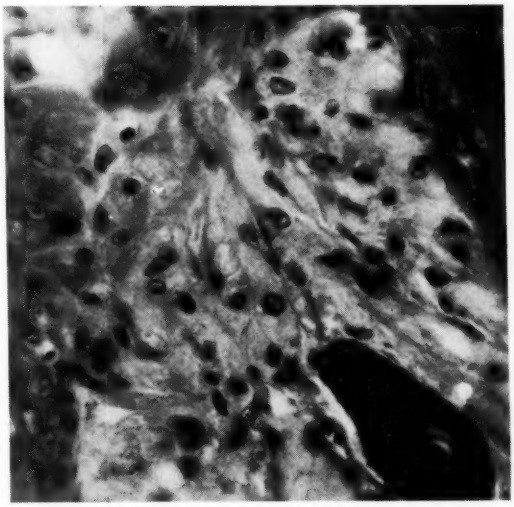

FIG. 1, CASE 1. Granular cell myoblastoma. X 412.

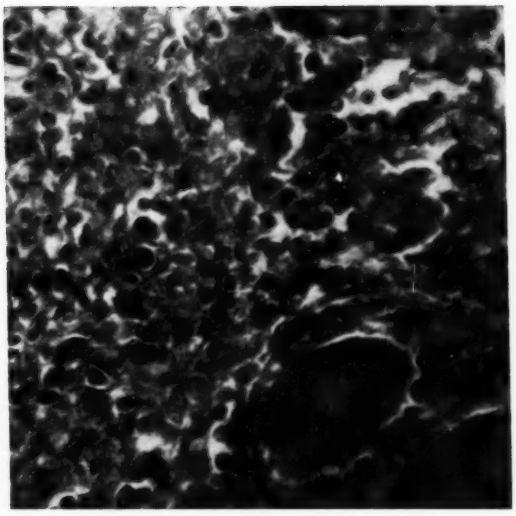

FIG. 2, CASE 2. Sarcoid showing typical giant cell. X 475.

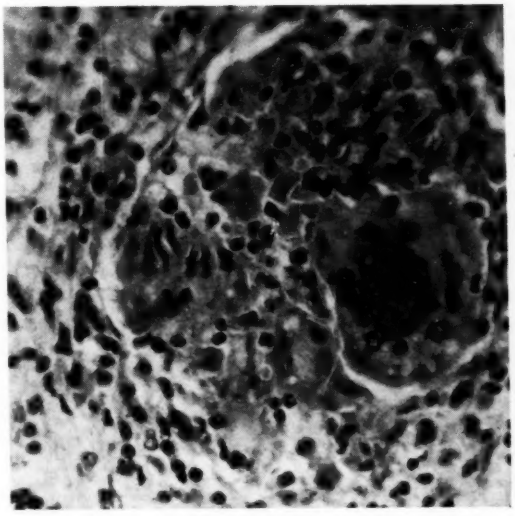

FIG. 3, CASE 6. Sarcoidosis. Note inclusions in giant cell. X 475.

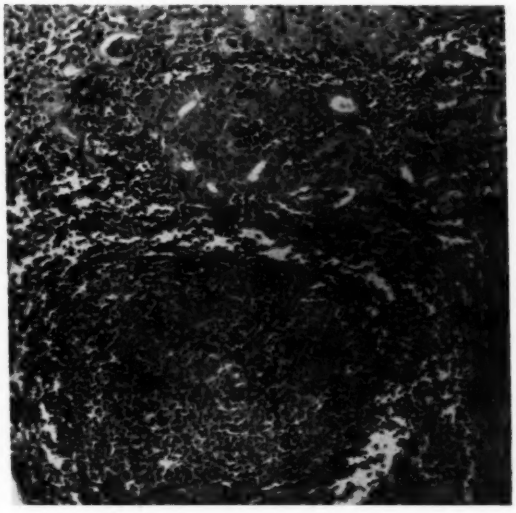

FIG. 4, CASE 7. Struma lymphomatosa. Heavy lymphoid infiltration and follicle formation. X 105.

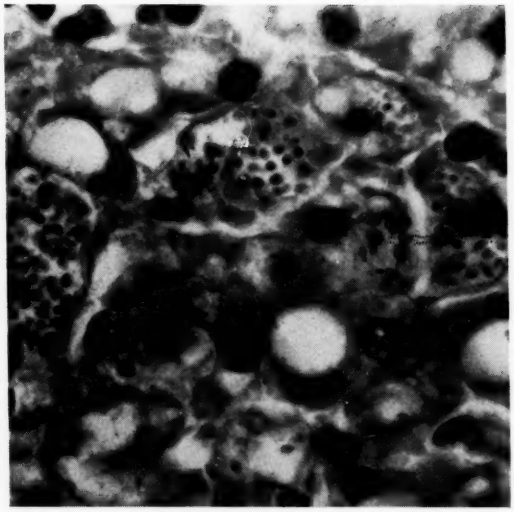

FIG. 5, CASE 8. Histoplasmosis. Note macrophages filled with Histoplasma capsulatum. X 960.

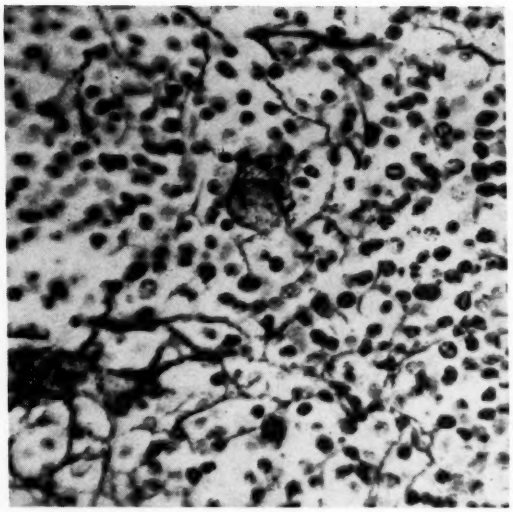

FIG. 6, CASE 9. Pharynx. Malignant lymphocytoma. Reticulin stain. X 475.

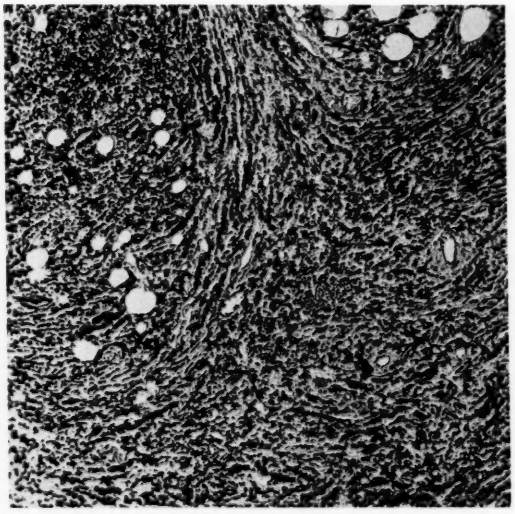

FIG. 7, CASE 12. Lymphosarcoma, lymphocytic type. Note fibrosis and infiltration into fat. X 105.

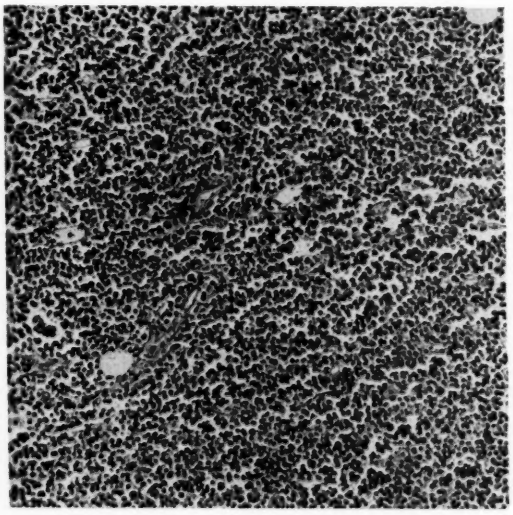

FIG. 8, CASE 13. Lymphosarcoma, lymphocytic type. Note diffuse pattern of small cells. X 105.

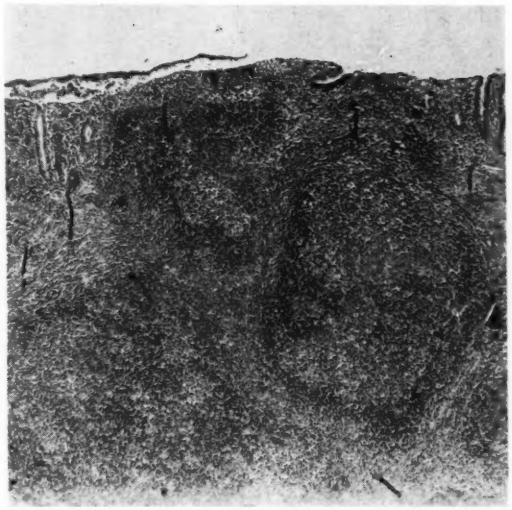

FIG. 9, CASE 14. Macrofollicular lymphoma. Note large germinal centers. X 30.

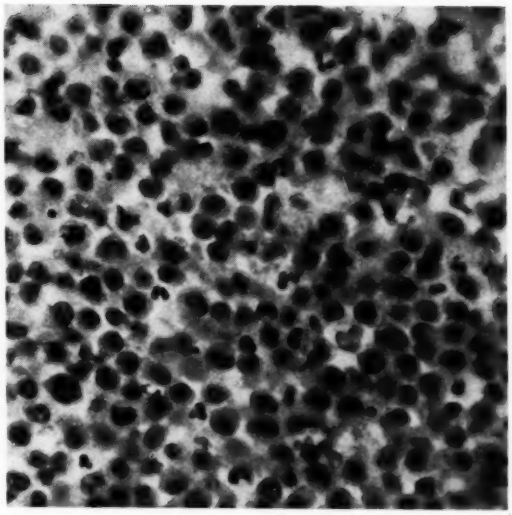

FIG. 10, CASE 15. Undifferentiated carcinoma. X 475.

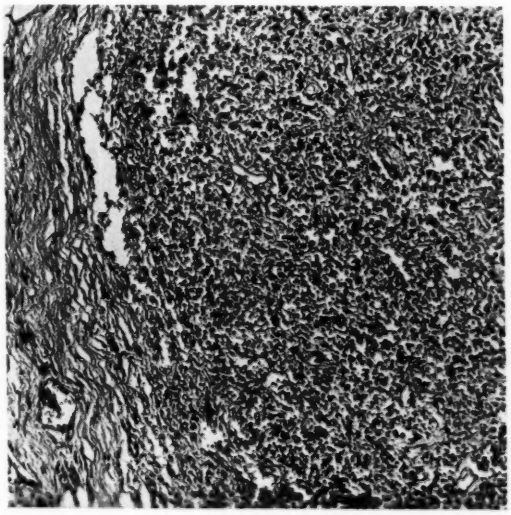

FIG. 11, CASE 16. Lymph node in Hodgkin's disease following treatment with nitrogen mustard. X 105.

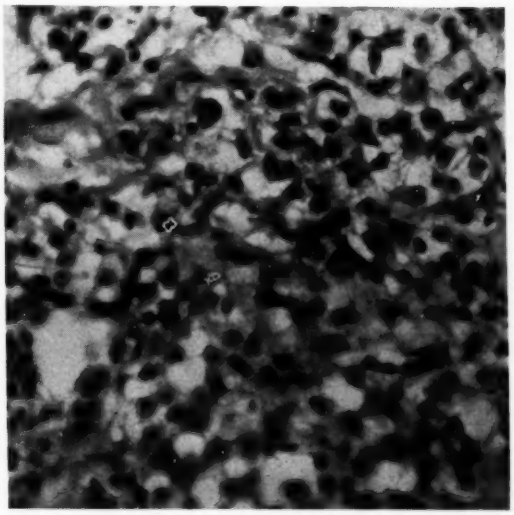

FIG. 12, CASE 17. Inflammatory granuloma of perineum. Eosinophils are numerous. X 475.

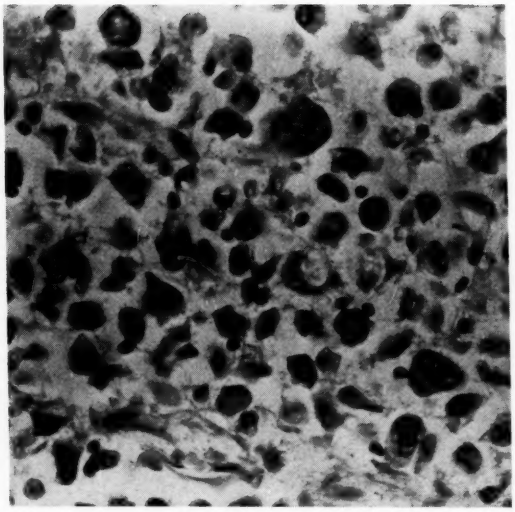

FIG. 13, CASE 18. Hodgkin's disease of small intestine. Note pleohistiocytes. X 475.

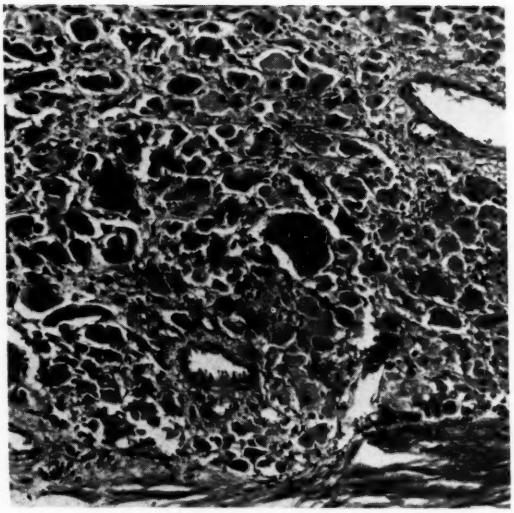

FIG. 14, CASE 19. Renal cell carcinoma. Note giant tumor cell in mitosis. X 105.

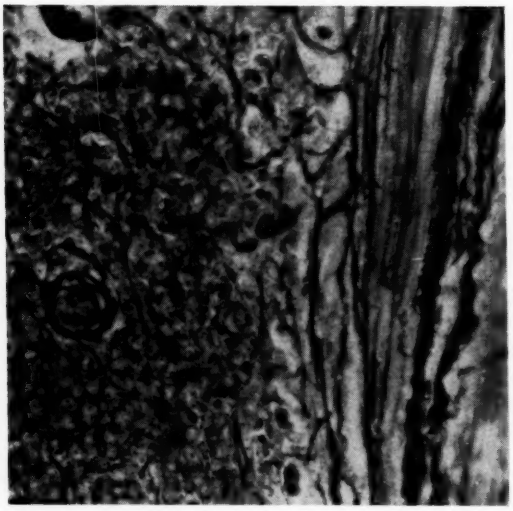

FIG. 15, CASE 20. Hodgkin's disease. Reticulin stain. X 475.

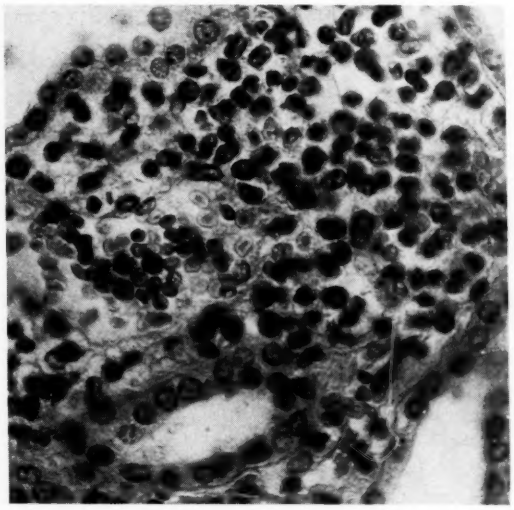

FIG. 16, CASE 22. Myelogenous leukemia of kidney. Note foci of immature myeloid elements. X 475.

Dr. Warren: There is some evidence that radiation will reduce the number of infecting organisms in a lesion, and it may account for the fact that there are relatively fewer organisms in this lesion than would ordinarily be expected. One of the things about the many cases of histoplasma is that the organisms sort of hit you in the eye. It is hard to find an area in which they are not present.

RETICULUM CELL SARCOMA

CASE 5.—Contributed by Dr. Warren.

Patient.—66 year old man.

Specimen.—Lymph node.

The specimen consists of a previously sectioned lymph node measuring approximately 4 by 2 by 1.5 cm. The cut surfaces are glistening, pale yellow-gray, and homogeneous.

Dr. Warren: This is a case of a 66 year old man with uneventful past history. There had been a fairly rapid development of enlargement of the lymph node. A biopsy specimen of the lymph node was removed. The node itself was glistening, the typical pale yellow-gray and homogenous surface that we have come to associate with a picture of most lymphoid tumors. Incidentally, in regard to biopsies in lymphoid tumors, it is worth while keeping in mind the tendency that surgeons frequently have to remove inguinal lymph nodes. I do not know of any lymph node from any part of the body that puts a tougher problem up to the pathologist than an inguinal lymph node. There is always just enough in the way of acute and chronic infections that have occurred in the drainage area of the inguinal nodes so that it represents not only whatever the present pathologic process may be but also the scars of any number of preexisting pathologic processes. One of the things that I always try to do working with the surgeons on the staff is to indicate to them that we will tackle an inguinal lymph node if we have to, but we would be a lot better off if they could get some other lymph node for us.

I wonder if Dr. Hartman would like to say what he feels from his combination of surgery and pathology with regard to this and related points on lymph node biopsies?

Dr. A. W. Hartman, San Antonio: I have heard you give the same advice so many times in the past that I am tempted to avoid the inguinal region. However, it is the easiest to get at, the least likely to leave a visible scar, and it is tempting.

Dr. Warren: Right! One place where it is important to resist temptation. (Laughter.)

Let us have a look at the slides for case 5. In this we see under low power a fairly evenly involved lymph node with all traces of normal structure gone, with a tendency of the cells to collect in clusters. At first glance when a pathologist sees a picture of this sort he thinks of the possibility of an undifferentiated carcinoma.

On this slide appears again the suggestion of an epithelial arrangement of the cells, the tendency to collection in clusters, with some normal stroma between, and yet the cells do not look right for epithelium. This sort of easy oozing of the cells over into adjacent areas is not at all characteristic of the epithelial type of structure.

Again in the higher power we see that the nuclei are not the characteristic epithelial nuclei with the prominent nucleoli, but rather they are somewhat vesicular; they have some aggregates of chromatin here and there but a diffuse powdery chromatin in much of the rest of the area. In some of the areas are pretty distinct, well marked, dense nuclei that this particular slide does not show as I see it from here, but one mitotic figure. This may be one too. I do not know whether there are others that you can see. There was a fair degree of mitotic activity in the section.

Here we have a lymph node. I might say that the capsule of this node showed capsular penetration. One of the important diagnostic points in lymphoid tumors is penetration of the capsule. I always feel a little shy about diagnosing a tumor as a lymphoid tumor unless there is a breaking through of the capsule and some degree of obliteration of the peripheral sinuses as well. One may find early enough involvement of the node so that this does not occur, but it occurs very very early in the development of any of the lymphoid tumors. Capsule penetration is not of much value in ruling out other conditions because in chronic inflammation or even in acute sepsis there can be penetration of the capsule.

Shall we see what the score is? Well, it is even right across the board: a lymphoblastic lymphosarcoma, a lymphosarcoma straight, and the reticulum cell sarcoma. I am going to throw in my lot with those who are thinking of the reticulum cell sarcoma, largely because of the epithelial-like clustering of the cells and the character of the nuclei.

Dr. M. Gerundo, Lubbock: Could this cell be of neural origin?

Dr. Warren: You have one of the neuro-sympathico-blastomas in mind. That would be an important thing to weigh. By and large there I usually expect somewhat more elongated cells with a somewhat conical or carrot-like shape. Another thing that was of importance here was that the reticulum stain, of which we did not project a slide, showed the characteristic diffuse reticulum of the reticulum cell sarcoma.

Dr. Leo Lowbeer, Tulsa, Okla.: Why would you not consider a lymphatic leukemia in the absence of

blood findings? We do not have any comments on the blood findings.

Dr. Warren: In this case the pathologist should probably think of an acute lymphatic leukemia, not a chronic lymphatic leukemia. Against that is the fact that did not show in the lantern slide: the capsular penetration. As a rule, in lymphatic leukemia you do not get capsular penetration; you get a stuffing of the gland with the immature lymphoblastic cells; but the capsular penetration does not occur. That is not a hard-and-fast rule, but it influences me somewhat. In order to bring out that point, which I hoped someone would bring out, we refrained from telling you that the blood count was normal and the differential normal. I think that it is always difficult from the slide alone to make a flat-footed diagnosis, particularly when one is dealing with an acute leukemia. It is always well to know what the peripheral blood shows in these cases. In fact, if one wants to do an ideal job of study, I think the model that the Army Institute of Pathology has laid down of the blood findings, the impression smear, marrow biopsy, and lymph node are all useful to have. From the standpoint of routine hospital pathology, it is difficult to get either the patient, the internist, or the surgeon to agree to as ideal a set-up as that.

In this particular tumor there was a follow-up with roentgen-ray therapy and a good initial response. The physician always keeps his fingers crossed in these cases because it is seldom that the patient will go more than a couple of years at best.

I know that Dr. Goforth has been interested in following these cases both from the hematologic standpoint and from the histologic, and has kept in good clinical touch with them as well. I wonder if he would care to make any comments on these groups of the reticulum sarcoma and the less differentiated lymphoid tumors in general?

Dr. J. L. Goforth, Dallas: My only comment is to reemphasize again the necessity for having collateral data along with the biopsy. I think you can certainly get trapped if you look at the biopsy specimen alone and extend the opinion too far.

Dr. Warren: Any further comments? Then we will move to case 6.

SARCOID

CASE 6.—Contributed by Dr. Warren.

Patient.—50 year old woman.

Specimen.—Spleen.

The specimen consists of a 405 Gm. spleen measuring 14.5 by 0.5 by 6 cm. The capsule is smooth and transparent except for a 4 cm. patch on the diaphragmatic surface where it is irregularly thickened by gray, translucent material having the appearance of tallow drops. There is a fluctuant 3 cm. cystic region just beneath the capsule and beneath the lower pole which on section contains fluid blood. On section, the surface is moderately firm and shows the usual follicles and trabeculae. Scattered through the pulp are smooth, shining, 0.1 cm. spherical nodules. Eleven of these pearly nodules are isolated.

Dr. Warren: This case involves a spleen, the description of which you have. In this one, in contrast to the earlier one that you saw (case 2), there was a scattering of smaller, more clearly defined nodules; and in your slides there were fewer nodules than in the other splenic case.

Here we see the spleen under low power with not much in the way of diffusely scattered granulomas present; fairly ordinary looking spleen.

Under higher power we see selected areas, the giant cells and the granulomatous lesion (fig. 3).

Here we see one of the giant cells with a clustering of the nuclei and distinction of the ring form that we earlier showed, and some nice examples of the spicules that frequently cluster together to make asteroid bodies.

In this case let us throw the diagnoses on now and see where we stand:

Chronic passive congestion. There may be some; I was not impressed by it particularly. There is a little diffuse fibrosis. There is a considerable enlargement of the spleen.

Healed tuberculosis. I would not think of that here because the giant cells are still present. If giant cells are present in a tuberculous lesion, I do not consider it as healed. I like to see either a complete replacement with hyalinized fibrotic tissue or calcification.

Hemangioma. No, I do not believe I would weigh that heavily.

Hodgkin's disease has to be thought of, but the giant cells are the giant cells of the foreign body type when you find them. We have not got the Reed-Sternberg cells that I like to see; we have not got the irregular fibrosis; we have not got the cellular pleomorphism. There is one point that is apparent in these sections: you cannot trust irregular nuclei in reticulo-endothelial cells. Reticulo-endothelial cells under a variety of stimuli will show large irregular nuclei with some clumping of the chromatin that will resemble, but not be identical with or characteristic of, the Reed-Sternberg type.

Myeloid metaplasia. I did not find myeloid metaplasia in my section. I did find a few eosinophils here and there and an occasional polymorphonuclear leukocyte, but those occur not infrequently in the spleen. Unless we get a nice formation of good hemopoietic islands I do not like to call it myeloid metaplasia.

Hyperplasia of the spleen? Yes, there is certainly a hyperplasia of the polyp involved here.

Apparently either we pulled a mean trick on you in not having enough of these sarcoid lesions scattered through the section, or they were so inconspicuous that you passed them up. I hope for our sake in playing the game fairly that that was the case. I purposely picked this out as a more or less confusing case because there were such rare lesions present in the section and I felt that it would be a good way of tracking down your powers of observation. It is a mean trick to play, but when you find the lesions they are so characteristic that I think it is well worth while.

Who would like to argue with this one?

Dr. M. Gerundo, Lubbock: Could you distinguish here from the splenomegaly a Gamna type? I have not seen the microscopic slides at all, but have seen it only there from the screen. I wonder whether there are similar Gamna nodules?

Dr. Warren: That is a good point. The question of the characteristics of the Gandy-Gamna nodules is sound since the spicular material that was present could correspond with it very well. However, the granulomatous character of the lesions, the other sections that showed the larger lesion, the ones that were described as being up to a millimeter in diameter, were so characteristic of the sarcoidal lesion that I had no hesitancy in ruling this out. I would certainly agree from the standpoint of what we have shown you on the lantern slide alone that that would have to be thought of. With the other evidence that we have, however, I think that we would be warranted in considering this as sarcoid, and the spicular material that is present as being the concretions that are associated with sarcoidosis.

I have no idea of what the etiologies of these inclusions in the case of sarcoid have been. There have been a number of speculations about them in the literature, but I have not seen any that are really satisfying.

Dr. Robert J. Morgan, Oklahoma City, Okla.: I would like to ask if you have seen the asteroid bodies in the skin.

Dr. Warren: Not in the skin, but in the sarcoidal lesions in the skin, in the giant cells of the sarcoidal lesions in the skin. They are not as frequent as they are in lymph nodes, but they are present from time to time. In fact they have helped me out of a tight place several times. One thing that is important in differentiation, particularly in the skin lesions from cutaneous tuberculosis, is to remember that getting a negative acid-fast stain on your section does not rule out tuberculosis, particularly if you use Zenker-fixed material. Anyone that is using Zenker-fixed material is fortunate if he gets 20 per cent of his sections to stain positively for the acid-fast organism. If he is using alcohol, formalin, or Bouin's solution he usually has a good deal higher proportion, but I happen to be using routine Zenker fixation in the general run of my surgical slides, and I am afraid to count in most instances on the occurrence or the absence of acid-fast organisms. It is an unreliable stain after Zenker's.

Dr. John J. Andujar, Fort Worth: I want to ask about the 3 cm. collection of blood which you describe beneath the capsule. Do you consider that a true hemangioma or simply a coincidental hematoma?

Dr. Warren: I was inclined to think of that as probably a simple coincidental hematoma. I do not believe it is a true hemangioma.

Incidentally, there is occasionally a hamartoma of the spleen which will result in considerable enlargement of the spleen without any obvious lesion being present, and in these days when there are more and more surgical splenectomies, it is well to have that in mind. If you are interested in veterinary pathology, it is worth keeping in mind that these hamartomas of the spleen are considerably more common in dogs than they are in the ordinary run of human material. If you find a diffuse distortion of the spleen with a considerable increase in size, which in section looks much like ordinary splenic tissue, with perhaps a considerably greater degree of vascularization than normal splenic tissue, the possibility of one of these hamartomas of the spleen ought to be carefully weighed.

CHRONIC THYROIDITIS HASHIMOTO

CASE 7.—Contributed by Dr. Warren.

Patient.—49 year old woman.

Specimen.—Thyroid gland.

The specimen consists of two lobes of thyroid weighing 25 Gm. and measuring 4 by 2.5 by 1 cm. and 4 by 3 by 1.5 cm. They are partially encapsulated, yellow-pink, and faintly lobulated. On section the surfaces are moist, gray-yellow, and homogeneous, with numerous pin-point yellow-brown areas.

Dr. Warren: I have to confess that I put in this case because of a special weakness that I have. I cannot keep away from the thyroid. It is one of my special hobbies.

Here we see a thyroid gland which shows a distinct diffuse involvement focally as well as diffusely with lymphoid tissue, some forming secondary nodules (fig. 4).

Under higher power, we see that the thyroid follicles are poor in colloid. That means that the hormone is being poured out about as fast as it is being

made because there is a fair inverse relationship between the outpouring of active thyroid hormone on the one hand and the storing of colloid on the other. As the output of hormone quiets down in the involution of a hyperplastic thyroid, there will be an increase in colloid. You note that in the masses of lymphoid tissue there is secondary nodule formation; there are really follicles present. You note that the thyroid cells are big. They are somewhat clear and have an acidophilic stain, the appearance that we associate with the Hürthle cell change.

A number of years ago the work of Marine demonstrated that these glassy, acidophilic, enlarged cells were crowded to the gills with mitochondria. The interpretation that has been placed upon them is that they represent a final effort of the cell to keep functioning under adverse circumstances. You see them in Riedel's struma; you see them in a variety of conditions where there are only a few thyroid cells left. I think that the picture is one of approaching exhaustion in an effort to maintain a high level of functioning activity.

Shall we see now what the diagnoses are on this one? Lymphadenoid goiter 1, and chronic thyroiditis Hashimoto 11. I would say that those were different names for the same thing and it looks as if everybody hit the nail on the head. I feel good in that everybody agrees with me.

Now in a Hashimoto: Can it be diagnosed clinically? We are fortunate in that Dr. Hartman not only was in the laboratory for a while but also worked in the Lahey Clinic surgically and saw a lot of thyroid cases there. Do you want to say whether you can diagnose a Hashimoto thyroid or not?

Dr. A. W. Hartman, San Antonio: I think you can approach that diagnosis; however, you are frequently wrong in that the Hashimoto is not necessarily symmetrical on both sides. Frequently it will involve only one lobe. It is considerably harder than you would expect in any normal thyroid and sometimes even harder than you find in the tumors. I think that you can get the feeling that a Hashimoto is present, or at least a chronic thyroiditis rather than a tumor, and certainly you can use your palpation to differentiate that from the Graves's disease.

While on this subject, I would like to ask you a question. We frequently see symptoms of hyperthyroidism in chronic thyroiditis and I have seen these cases subside and actually the gland improved on propyl-thiouracil. I wonder if that experience has been had in Boston?

Dr. Warren: You have a sound point. I think there was a good deal of evidence that most of the cases of Hashimoto have had an antecedent history of hyperthyroidism. As I recall it, when Dr. Beck was working at the Memorial Hospital in Worcester, he had a long-time follow-up of one of these cases. Is that right, Dr. Beck?

Dr. James S. Beck, Tuscaloosa, Ala.: This patient was operated on and a gland weighing about 30 or 40 Gm., as I remember it, was removed. In it we found the large Hürthle cells and I thought that we might be dealing with Hürthle cell tumor. However, scattered throughout the entire gland were these large areas of lymphoid tissue with scattered nests of better looking thyroid gland tissue here and there. I do not know what the outcome is as I have had no connection with the case for about six years.

Dr. Warren: The problem of the use of the thiouracil derivatives in Hashimoto cases I cannot answer very well at present. I do not think we have had enough cases that have been followed up as yet to have a final answer. I would not hesitate to use propyl-thiouracil in such a case, but I do not know what the long-range outcome will be.

Now a word about the gross differential diagnosis: In the first place, while it is still in the patient's neck, in the perfectly typical case of the Hashimoto form of the thyroiditis, there is a fairly easy finding in the neck in the typical case. As Dr. Hartman mentioned, the gland is enlarged and moderately firm; it is not fixed. It is definitely outlined and it is smooth in outline. It is usually symmetrical but sometimes is unilateral. When it is unilateral, however, it involves the entire lobe. It is rare to find it restricted to just a portion of a lobe, and I do not believe you could diagnose it clinically under those conditions. There is usually present either some evidence of hyperthyroidism or some evidence of past hyperthyroidism.

From the standpoint of differential diagnosis, the pathologist has to consider the diffuse physiologic enlargement of the postadolescent goiter, the diffuse enlargement that occurs in the course of pregnancy, and some forms of hyperthyroidism in the stage of remission where the gland is still large, still somewhat firm, but the functional activity has fallen and there is enough colloid present to give a fair degree of firmness. Once it is out of the neck and on the table or in the pathologist's hand, the diagnosis is easy because the capsule is maintained. The texture on section is fairly homogeneous and the full blown specimen looks much more like a section of a lymph node than it does a thyroid tissue. There may be here and there a few streaks and mottled patches of thyroid tissue, but in general it looks much more like a big, firm, juicy lymph node than it looks like anything else.

Dr. M. D. Blackburn, Jr., Dallas: I would like to know if you could briefly give chronologically the

development of Hashimoto's and Riedel's strumas, and if you think there is any relationship between the two?

Dr. Warren: That is really rough to answer. There are those who believe that Riedel's struma usually arises on the basis of an old Hashimoto, or it goes through the phase of Hashimoto. There are always some lymphoid cells present in the typical Riedel's struma along with the fibrosis; along with the atrophy of the thyroid tissue there is some lymphoid infiltration. We have had cases of Hashimoto that had initially a partial thyroidectomy, the reason for that being that if a fairly complete thyroidectomy is done in these cases, the patient will be placed permanently on thyroid extract. If just the isthmus is taken out, it is possible to get away from any obstructive symptoms that may develop and yet permit the functioning portion of the gland to remain. There have been enough of those cases that have gone a number of years without the development of Riedel's struma to make me feel that the Hashimoto condition is a lesion in its own right which usually stays and does not turn into Riedel's.

From the other standpoint, in going over the history of the typical Riedel's struma we frequently find a history of acute thyroiditis at some time in the past. One of the best cases of Riedel's struma that I saw was in a medical friend of mine in New York. He had a stormy course of acute thyroiditis, with cellulitis of the neck. After a rough time he recovered smoothly and at the end of three years cropped up with a perfectly typical Riedel's struma.

In some of the Riedel's cases there is no good story of antecedent acute thyroiditis. Actually, although in the literature there has been almost equal emphasis placed on these lesions, in my experience —which is now, thanks to Dr. Lahey and his group, something over 26,000 thyroids—there is a much rarer incidence of Riedel's than of Hashimoto's struma. There have been a bare handful of Riedel's and a good assortment of Hashimoto cases. I think that if you will check back over your own experience, you will find that you have seen a number of Hashimoto's for each Riedel's struma that you have seen.

Dr. J. L. Goforth, Dallas: Have you seen Hashimoto's lesions in men?

Dr. Warren: Yes, I have; it is not nearly as frequent as in women; however, we must remember that all pathologic conditions of the thyroid, with the exception of endemic goiter, are essentially a female prerogative.

Speaking of endemic goiter and looking at these neat and trim Army personnel here today, with the sensible type of blouses that they now wear, I am reminded of the experience I had at the camp at American Lake, Fort Lewis, outside Seattle, during the war in 1918. Recruits from the Northwest there were the sloppiest soldiers I think I ever laid eyes on because they came fram an endemic thyroid region. You remember the old style blouse had a tight collar. If the recruits wore collars that were large enough so that they could be hooked, the sleeves and the blouse level came down to their knees. If they had a blouse that would fit otherwise, they had about a 2-inch gap in their collars. That was in the days before Marine's work had been widely appreciated and before the use of iodine in controlling the various forms of endemic goiter had been brought about. Now that would be a rare happening. Endemic goiter is practically the only form of thyroid disease where there is not a sex predilection. Aside from that condition, thyroid disease runs almost uniformly somewhere between 7:1 and 9:1 as the prerogative of the female sex.

Another striking sex differential that is in favor of women in this instance is carcinoma of the skin of the ear. As I look around at the attractive ladies here I notice that practically every one of them keeps the upper part of her ears tucked in under her hair, which is a wise thing to do because the part of the ear that develops carcinoma is usually the upper part of the pinna. In 9 cases out of 10 carcinoma of the ear is in males who wave their ears out in the breeze. They get them frostbitten; they get them sunburned; they get them exposed to the weather; and they get this high frequency of carcinoma of the ear.

You remember the experience of Dr. Lacassagne who made extensive studies of skin tumors of the face in France. There the women do the work out in the fields, and there most of the carcinomas of the skin of the upper face are in women. But, because of that same habit of keeping their ears covered up, they do not have carcinomas on their ears.

Any other points about Hashimoto?

Dr. M. Gerundo, Lubbock: I want to make an observation that cancer of the skin is extremely rare among the Japanese.

Dr. Warren: Yes. Incidentally, the Hashimoto name does not imply a Japanese origin for the disease. Hashimoto was working in Berlin in 1901. The application of his name does not imply its incidence in Japan. Incidentally, in the autopsies that Colonel DeCoursey and I did on the atomic bomb victims at Nagasaki, we did not see thyroid disorders in the group. I think probably the iodine rich diet due to the nearness to the seacoast in the Japanese islands and the high proportion of fish in the diet would keep their iodine levels up.

One cannot trust geography entirely in relation to

thyroid disease. I was much puzzled when Dr. Ratcliffe, the pathologist of the Philadelphia zoo, told me that in their coyotes, wolves, and foxes colloid goiter was occurring. I could not understand why, as close to the seacoast as Philadelphia is, the animals in the zoo should be getting this form of iodine lack of thyroid change. In checking up, however, we found that they were eating a prepared food which came from Ohio. When they were put on local food, the problem ceased.

There is another interesting point: One of the early experiments on the neural origin of hyperthyroidism—Walter Cannon's initial experiments in cats—got badly stymied for a while because in the first stage of his experiments the cats were being fed salt-water fish as the cheapest form of food and they were getting a rich iodine supply. They were changed to horse meat, which originated at that time from Oregon and the inland Columbia basin where iodine is scarce, and Cannon could not get reproducible results until he checked back and found that this difference in feed and difference in iodine intake had occurred.

Another minor point: It might interest you to know that I learned from Dr. Donaldson, the professor of aquatic biology at the University of Washington, that the predatory animals which habituate the banks of the Columbia and the Snake Rivers do not get thyroid abnormalities while the ones which are in the plains adjacent do suffer thyroid grief. The reason is that the salmon runs are sufficiently great to bring in an adequate iodine supply for the animals along the river banks that eat the dead and dying fish, whereas those which do not get this source of iodine get the endemic form of colloid goiter.

HISTOPLASMOSIS

CASE 8.—Contributed by Dr. A. O. Severance, Baptist Memorial Hospital, San Antonio.

Patient.—5 month old girl.

Specimen.—Spleen and liver.

The child was one of adopted twins who had shown only slight dietary difficulties until the present illness; she had not eaten as well as her twin brother or gained weight as rapidly. Ten days prior to admission she developed "cold" and once had bloody sputum. She suddenly had a paroxysm of coughing, became cyanotic, and presented a picture of pharyngeal obstruction. There was frothy, bloody sputum. The spleen was greatly enlarged. Roentgenograms of the lungs and long bones were normal. There was moderate anemia. The white blood cell count was 10,100, with 56 per cent neutrophils, 25 per cent lymphocytes, and 2 per cent myelocytes. At autopsy, the spleen was found to be four times normal size, with a beefy red cut surface and everted edge. The liver was also enlarged and on section appeared "nutmeg." The lungs appeared consolidated and showed numerous hemorrhagic nodules throughout.

Dr. Warren: This is a case that I think Dr. Severance could do a much better job with than I. Would you come up, Dr. Severance, and give the picture?

Dr. A. O. Severance, San Antonio: In this 5 month old baby the material at autopsy was prepared by a general practitioner in a little town about 125 miles from here and then sent to me for study. The features of the slides I think are easily brought out by the photographs that Major Stansell has made. Shall we see those now?

Here are large cells filled with these round bodies (fig. 5) with a little clear zone around them; they stain dark blue in the hematoxylin stain. The interesting thing to me was that these cells were so abundant throughout the sections.

One other thing which you do not have available in your slides is this fact: In a lymph node in the peribronchial region there were present tuberculous-like lesions with giant cells of the Langhans' type and epithelioid cells; and in both the Langhans' giant cell and in the epithelioid cell were these same spore-like organisms.

The differential diagnosis, as it occurred to me, involved trying to separate histoplasmosis in this 5 month old child, born in this country, from some form of leishmaniasis. The clinician who did the autopsy also sent me blood films made from heart blood at autopsy and also from the bone marrow. In Wright stains on those blood films we saw the large phagocytic cells, and they contained these same organisms. I could find only a single large red-staining organism in these little bodies. I did not find the so-called kinetoplasts. I had Major Bayliss looking at these with me. He did not think that it was leishmaniasis either and we did not see why it should occur in this country, so that forced us back to the diagnosis of histoplasmosis. I felt with that lesion in the lymph node in the peribronchial region that we had a little more support for that diagnosis.

Dr. Warren: I think this is particularly helpful in bringing out clearly some of the high magnification pictures that we did not have available in the earlier case we had for consideration.

Dr. Severance: Let us see the score board; that indicates that the diagnosis of histoplasmosis wins hands down.

The next lantern slide demonstrates the numerous small bodies within the reticulo-endothelial cells of the liver, and we feel that these are organisms of histoplasmosis.

Dr. Warren: We appreciate your demonstration of this case, Dr. Severance. This case is particularly beautiful because the parasites hit you in the eye here, and they are widely distributed in the liver, particularly in this last slide, showing up well their storage in the reticulo-endothelial cells. I think there

is hardly a reticulo-endothelial cell in the whole section of liver I looked at that did not have these organisms stored in it.

This is a much rarer disease in the New England area than it is down here, or else we are much poorer at recognizing it than you are here. The majority of cases that have been reported, as I recall it, have come from the South and Southwest. Is that not the case, Colonel DeCoursey?

Col. Elbert DeCoursey, Brooke General Hospital, Fort Sam Houston: It is interesting that this disease was first described by Darling in Panama when he was looking for kala-azar, and it has been difficult to find a case since. Dr. Elkins is down there now. He says he must keep up Dr. Darling's reputation and find another case; the same thing with Schlatter's disease. There are about 100 cases of histoplasmosis in the literature, many of them from the Mississippi central valley. Somewhere between 25 and 35 per cent I believe are in children, and about 20 per cent in less than 15 months old infants. This case is in a young child also.

Dr. Dudley Jackson, Sr., San Antonio: It may be of interest that in reporting my 1 case we first thought the condition was tuberculosis of the tongue. We used some radium on it and it did not respond. In the second biopsy, after the radium had been used, Dr. Severance was able to make the diagnosis clearly. The patient was also given large doses of penicillin in the beginning with no effect.

Dr. Warren: Any other comments on this case? How frequently is it seen in the Memphis and Tennessee valley area, Dr. Tribby?

Dr. William W. Tribby, Memphis, Tenn.: I have not had any cases of my own, but Dr. Moss at St. Joseph's Hospital has had 1 case during the past year; that is the only one I know of occurring in that vicinity. They may have some at the University of Tennessee.

Dr. Warren: Is there any sound evidence on what the portal of entry is in these cases, Dr. Lowbeer?

Dr. Leo Lowbeer, Tulsa, Okla.: I do not know about the portal of entry; however, there is a paper published by Dr. Ferringer, from the University of Oklahoma, in which he maintains that the disease may be transmitted from dogs, in which case the portal of entry would be the mouth.

Dr. R. H. Rigdon, Galveston: There were several cases reported many years ago from Memphis; I recall one in 1939 in a baby like this. There is a high percentage of positive skin tests in Arkansas, Tennessee, and Missouri. We have been on the watch for these cases at autopsy but we have missed them if they have occurred.

What has been your experience with the frequency of demonstrating the organism in such large numbers as this in the routine cases?

Dr. Warren: I think there are few cases I have seen that show as large numbers and as many cells involved as this one of Dr. Severance. This is an excellent case.

Dr. J. F. Pilcher, Corpus Christi: I wonder if the twin has been ill at all or has been skin tested for histoplasmin since this other twin died?

Dr. Warren: I think that Dr. Severance's signals say that that has not occurred as yet.

Audience Member: Can you discuss the possible relation of histoplasmosis to calcified nodules of the lung?

Dr. Warren: I am sorry; I have no experience in the relation to calcified nodules. My general assumption when I find a calcified nodule of the lung is that in view of the prevalence of pulmonary tuberculosis, it is probably a healed lesion of pulmonary tuberculosis. We know that other lesions can heal with calcification. There is some evidence that a fair number of the fungal infections come in by the respiratory pathway. However, in this particular field my experience is not satisfactory and I have no opinion that is worth anything on it.

MALIGNANT LYMPHOCYTOMA

CASE 9.—Contributed by Dr. A. O. Severance, Baptist Memorial Hospital, San Antonio.

Patient.—75 year old white man.

Specimen.—Mass from hypopharynx.

The patient had a growth in the throat of from three to six months' duration. The mass partially interfered with swallowing, speech, and, at times, respiration. The mass arose from the left pyriform fossa and extended up the left pharyngeal wall almost to the base of the left tonsil. The Kline test was negative; the white blood cell count was 11,850, with normal differential.

The specimen consists of a soft grayish-red mass measuring 3 by 2 by 1.5 cm., covered on one surface by normal appearing mucosa.

Dr. Warren: The nearest slide gives some of the low power view of the tissue, a similar low power view of the epithelium coursing along here, and a reticulum stain with the individual cells clearly brought out. This impresses me particularly from the standpoint of the infiltration of the tissue that has occurred. This is the equivalent of a good infiltration of the capsule, and I would say that the excess of lymphoid tissue out into this area, of a diffuse character such as this, is good evidence that we are dealing with one of the lymphoid tumors.

In this section, which gives the clearest picture of the individual cells, we see that there is an irregular production of reticulum which is disseminated through the substance of the cells, and that the in-

dividual cells are fairly small and compact, with well defined nuclei and not much mitotic activity (fig. 6).

Let us see what the score is on this case: lymphocytoma 1, lymphosarcoma 6, lympho-epithelioma 2, and lymphoblastic type of lymphosarcoma 4.

Everyone, with the exception of two, is agreed on the essential lymphoid character of the tumor. First, a word about the lympho-epithelioma. I would rule that out in this case because in the lympho-epithelioma one tends to have closer packed cells than we have here, rather more elongated cells, with a more definitely epithelial character of the nucleus. Here, I think practically all of the cells are distinctly lymphoid in character. Perhaps Dr. Severance would tell us whether that held true for the entire specimen as well.

Dr. A. O. Severance, San Antonio: The slide is representative of the entire picture.

Dr. Warren: I judge that in this case also there was no abnormality of the blood count. For white blood cells I consider 11,850 as upper normal, but not significantly elevated. From this section I could not tell whether we were dealing with a lymphatic leukemia or not. I have seen occasionally in the enlargements of the tonsil that occur in lymphatic leukemia a picture similar to the one here. I wonder whether the diagnosis of lympho-epithelioma arose from the fact that the pathologists noted the mass arose from the left pyriform fossa. By and large, the pyriform fossa is a good place from which lympho-epitheliomas come. In this instance, however, I think we are dealing with a straight lymphoid tumor and not one in which the epithelial elements have to be considered. I would be inclined in this instance, and in view of the history of a localized tumor and the normal blood count, to say that this is a lymphoid tumor of a well differentiated type, the type I would call a malignant lymphocytoma, or as it is frequently spoken of, a malignant lymphoma, which is the best differentiated form of the lymphoid tumors. I think that is essentially in agreement with the general score on the board.

LYMPHOSARCOMA

CASE 10.—Contributed by Dr. A. O. Severance, Baptist Memorial Hospital, San Antonio.

Patient.—5 year old white boy.

Specimen.—Ileum.

The patient presented an eight week history of nausea, vomiting, and diarrhea, followed later by constipation. Examination after barium by mouth revealed passage to the region of the cecum, with little entering the cecum. After a barium enema the cecum was visualized normally but the barium did not enter the ileum. At operation the ileum was found to be intussuscepted into the cecum, with gangrene of the bowel wall. The wall of the involved ileum was edematous and the lumen was obliterated.

Dr. Warren: Case 10 is a lesion of, and this particular section comes from, the ileum.

In this slide we see, first, the mucosa of the ileum, with ulceration. One of the interesting things when these lymphoid tumors ulcerate is that there are usually no polymorphonuclear leukocytes present, and rarely much if any fibrin. In some ways, at first glance, the pathologist can be fooled by the ulcerated Peyer's patch of typhoid fever. I do not suppose that any of you run across pathologic specimens of typhoid fever nowadays, but I was brought up in the laboratory of Dr. Mallory, who gave the first adequate histologic description of the lesions of typhoid, and had drilled into me the fact that when there was an ulcerated Peyer's patch without any polymorphonuclears along the edge, typhoid fever was a good thing to pick off. Unless we remember that there can be lesions of typhoid, it is easy to overlook that disease. In this country, of course, we have it well under control. Those of us who were in Japan awhile saw a good scattering of cases there, although I understand now from the last report from Supreme Commander Allied Powers that even there the "happy hunting ground" for pathologists has been spoiled. They have gotten typhoid fairly well under control. The advances of public health are not always in the best interests of the pathologists seeking abundant and curious material. (Laughter.)

In this slide we have an ulcerated lesion. We note that there is a diffuse and irregular fibrosis. We note that there is separation of smooth muscle fibers and that there is no definite structure. This is not a granulomatous process. This is a diffuse infiltration of the cells.

When we turn to the higher power, we see that those cells are singularly uniform. We see that they are arranged sometimes, if there are long strands, in columns, but in general they are independent cells, sharply defined, not adherent to one another, not integrated with one another, not carrying a definite stroma, but likely simply infiltrating the preexisting stroma.

That same holds true in this section and in the good demonstration of the larger scale here, where we see that the cells are fairly large, that they have more cytoplasm than the adult lymphocytes, that there is an occasional mitotic figure, that they are fairly uniform, but in general less mature than the common run of adult lymphoid tumors. I think that all of us would agree from this picture that we are dealing with a tumor in the lymphoid group, even though it comes from the ileum.

Let us see what the score is: lymphoblastoma (I imagine that is used in the general sense) 1, reticulum cell sarcoma 2, lymphosarcoma 3, hemangioma 1, and neuroblastoma 1.

From the standpoint of the advocates of the reticulum cell sarcoma, I think these cells are a bit too well differentiated. They are pretty much of the lymphoblastic type. The predominant cells are small. Nowhere did I see tendency to syncytial arrangement, and in the general detail of the nuclei, which you could see somewhat in one of the slides, I felt there was not the fine powdery diffusion of chromatin that one is apt to get in the nucleus of the reticulum cell, aside from the major areas of concentration in what looks like nucleoli. I do not believe I would want to ride along with reticulum cell sarcoma in this case; lymphosarcoma is my own leaning.

In hemangioma I think one would have to account for the cause of so heavy a lymphoid infiltration and a lymphoid infiltration that not only occupied normal spatial relationship but which infiltrated and separated normal cells from one another.

Neuroblastoma I would be inclined again to rule out on the basis of the rounded character of most of the cells, the absence of any roset or pseudo-roset formation, and the absence of the elongated, somewhat carrot-shaped cells that we usually see, at least in some areas, in neoplastic tumors. That is one of the difficult differential diagnoses.

Any comments, questions, or discussions on this tumor?

Dr. William W. Tribby, Memphis, Tenn.: I would like to ask if in general the lymphocytomas respond to roentgen-ray treatment less well than the lymphosarcomas?

Dr. Warren: As I have seen them, I would say that the response is about the same. The roentgenologist of Brooke General Hospital is still with us. Would you care to comment, Colonel Ackerman, as to whether there is any significant difference in the response to radiation therapy of the well differentiated lymphoid tumor and the lymphosarcoma; the malignant lymphocytoma on the one hand, and the lymphosarcoma on the other?

Lt. Col. Alfred Ackerman, Brooke General Hospital, Fort Sam Houston: I do not believe that the final response is much different. One thing, perhaps, which is worth while emphasizing is that contrary to the old opinion that those tumors require relatively low doses, we should give them rather high doses initially because the majority of the longest periods of remissions are obtained with the highest treatments when the tumors are first treated with radiation. All subsequent treatments respond less adequately, and as you know, they may be improved somewhat by a combination of radiation and nitrogen mustard. Relatively speaking, they do fall in one pattern and their behavior is similar.

Dr. Warren: You have had a good deal of experience with them, Dr. Goforth. Would you go along with that?

Dr. J. L. Goforth, Dallas: Yes, I would. I would like to ask this question in that connection. In your experience, is there any way that one can study a slide of this type and formulate an idea as to whether or not the condition is going to transform itself into a terminal leukemia or into one of the other forms of lymphoblastic diseases?

Dr. Warren: I have not been successful in that. I have tried hard and have been completely without success.

One thing with regard to that point on therapy is worth emphasizing. It certainly is true that while one gets regression with slight doses of radiation, if one is trying for a cure or a long-term remission, heavy dosage is certainly indicated, and it holds with the lymphoid tumors as with the other types of tumors which are radiated. The first time that you hit them is your best chance. You may aid in building up resistance to radiation by too light radiation over prolonged periods.

LYMPHOSARCOMA

CASE 11.—Contributed by Dr. D. A. Todd, Nix Hospital Laboratories, San Antonio.

Patient.—51 year old white man.

Specimen.—Biopsy sample from the rectal area.

The patient had had hemorrhoids, with protrusion and intermittent bleeding, for sixteen years. A generalized lymphadenopathy which he had had for many years was attributed to malaria during World War I. Examination revealed a generalized lymph node enlargement which was firm and in only a few areas appeared attached to the skin. The recto-sigmoid area showed a lining thick, firm, and ulcerated. There were few internal hemorrhoids.

Dr. Warren: There is one thing that I would like to mention with regard to the involvement of the small intestine by tumors. If when you get a metastasis, particularly an ulcerating metastasis or a metastasis on the mucosal side of the small intestine, it is almost certain to be either a lymphoma, one of the fixed tissue masses of a lymphatic leukemia or a monocytic leukemia, or a malignant melanoma. It is extraordinarily rare to have metastases of the other types of tumor in that locality. I have, with the help of Dr. Meissner, Dr. Gates, and others of my staff, made a fair analysis of the distribution of metastatic lesions, and so far as the small intestine is concerned, those three types of lesions run well ahead of all the others.

Case 11 is Dr. Todd's case, and it has a somewhat confusing history.

This slide is a biopsy specimen of the rectal region. There is a history of hemorrhoids for sixteen years and generalized lymphadenopathy for many years, attributed to malaria in World War I. I would like to

ask Colonel DeCoursey how often malaria gives rise to long continued lymphadenopathy? Have you ever seen a case of malaria wind up in generalized lymphadenopathy?

Col. Elbert DeCoursey, Brooke General Hospital, Fort Sam Houston: The only lymphadenopathy that we saw in Panama—if you call splenomegaly a lymphadenopathy...

Dr. Warren: No.

Colonel DeCoursey: Then if we had it, I missed it. I did not see any.

Dr. Warren: I do not think there is any relationship from all of the evidence that I have been able to pick up. Splenomegaly has a relationship, as a rule, because that is one of the organs of the blood side of the blood-lymphatic system, but lymph node enlargement I do not believe is significantly related to malarial infection. Of course, there is no statement here that it is. Patients attribute all sorts of things, but it is well to bring out that point clearly.

One of the interesting things here is that in some places the lymph nodes appeared fixed to the skin. That means usually, if we are dealing with a lymphoid tumor, an advanced stage, and I have come to the conclusion that the fixation of lymph nodes to the skin in the course of a lymphoma is just as sour a prognosis as a fixation of carcinoma of the breast, say, to the skin. When you find that, you usually find not only a case that is going to do badly but which is close to the end of its life span. It is rare, when you get the nodes so heavily joined that there is fixation to overlying skin, that you will get a satisfactory outcome.

Now, let us see how the section looks.

In this slide we see first a residual film of the colon taken after the bulk of the barium has passed; the radiologist was hunting for polypoid lesions. We see here that there is a dilatation of a fair portion of the sigmoid with some retention, but not much. I understand that it is the opinion of radiologists that a film of this type would mean little indeed. I am showing it chiefly from the standpoint of saying that with the exception of the differential diagnosis of diverticulosis and diverticulitis, with the exception of getting the frankly polypoid lesions by the contrast mucosal technique, there is relatively little chance of picking up a rectal or a sigmoidal lesion. By and large, I would say that the use of a roentgenogram in trying to determine the presence or absence of any lesion within reach of the sigmoidoscope is a waste of time both for the patient, the doctor in charge, and the radiologist who takes the picture. There may be some conditions under which that does not hold true, but so far as suspicion of tumor is concerned, and within reach of the sigmoidoscope, I have yet to know of an instance in which the roentgen ray turned up anything that the direct examination did not, or that turned up nearly as good evidence as the direct examination itself brought out. Perhaps we ought to take a second and allow our radiologic friends to refute me on this point if I am wrong. No dissenting voice, so we will let it ride.

On this slide we have a reticulum stain on the tumor, which shows that there is a little increase of finely divided reticulum fibers scattered in through. Incidentally, I ought to pay my iconoclastic respects here a little bit to the reticulum fibers. I rarely do a reticulum stain in order to determine whether I am dealing with a reticulum cell sarcoma or not. I realize that that is perhaps not in accord with best practice, but I have found by and large that I learn so little more from the reticulum stain that it is not worth while, and I have made up my mind primarily on the basis of the histologic picture as presented. I would not hesitate to make a diagnosis of reticulum cell sarcoma in the absence of the reticulum fibers. I think one unfortunate connotation of the emphasis on reticulin fibers in connection with the reticulum cell sarcoma is that in the least differentiated, the most malignant form, where you still have large syncytial masses, you may have virtually no formation of reticulin at all. It is not an essential for the diagnosis to demonstrate reticulin fibers. I use the reticulin fibers chiefly in helping me differentiate between lymphoid tumors and epithelial tumors because the clusters of epithelial cells in most instances are surrounded by a rim or net of reticulin, and it is never as clear-cut in the lymphoid tumors as it is in surrounding the little nests of the epithelial tumors.

In this next slide we notice that the cells are much the same type. There is a diffuse infiltration outrunning the main tumor mass, and here and there large hyperplastic reticulo-endothelial cells are scattered through. This is seen better here, and one can almost think of this as representing a peripheral sinus along here. The equivalent of capsule, although this is not definitely a lymph nodal structure here, and the infiltration of the fat, are well shown. Here is the detailed cytologic structure, and we see cells that are in the lymphoblast group, I believe, well recognizable as being in the lymphoid series, but certainly not adult lymphocytes, with still some cytoplasm, with a more vesicular type of nucleus than the compact one that we have in the adult lymphocyte.

Let us see what the score is on this one: lymphocytoma 4, giant follicular lymphoma 6, Hodgkin's disease 1, lymphosarcoma 1, and mycosis fungoides 1.

We are all thinking of lymphoid tumors here.

Lymphocytoma, I think, is certainly in the running. I believe that the cells are a bit less differentiated. As to giant follicular lymphoma, there certainly are giant follicles here. Although the prevailing cell is of the lymphosarcoma type, its arrangement is still in follicular fashion, with the suggestion of lymphoid structures still present. Hodgkin's disease I would not want to consider because although there are irregular nuclei in the reticular cells that were present, I do not think these are neoplastic nuclei. They look more like hyperplastic reticulo-endothelial cells than neoplastic ones. Lymphosarcoma is certainly in the running. Mycosis fungoides will hinge on terminology. Properly that is mainly applied to cutaneous tumors, and my feeling is that the great bulk of cases diagnosed as mycosis fungoides, pathologically, are simply cutaneous manifestations of Hodgkin's disease. There are some cases in which only the skin is involved and in which perhaps mycosis fungoides is a proper lesion. I would not want to consider it in this particular instance although it falls in the general group of the lymphoid tumors.

Now let us see what the discussion is. Perhaps we might ask Dr. Stout what his and Dr. Todd's diagnosis was on this particular case.

Dr. B. F. Stout, San Antonio: I do not know what Dr. Todd's diagnosis was, but my own was lymphosarcoma, small cell type.

Dr. Warren: This is a tumor, in light of the long history, in which you would lean toward its being one of the better differentiated forms. In view of the fact that there is a good deal of follicle formation and a long history, the possibility of one of the macrofollicular types would have to be taken into consideration. Without having a section from the lymph node, I would not want to say, because I have seen instances where the picture in the visceral involvement of the macrofollicular type was not carried over, even though it was apparent in the lymph node itself.

Any questions or suggestions with regard to this case?

Dr. Elsie McPeak, Houston: Do you think that this history of some difficulty of hemorrhoids may have been related to the process of this disease?

Dr. Warren: I think it is a possibility. If so, we would almost certainly be forced to put it in the macrofollicular type. I cannot imagine a sixteen year history with the ordinary run of lymphosarcoma or malignant lymphocytoma. Usually when you get a history as long as that, you have to think of either a macrofollicular lymphoma or Hodgkin's disease. I know of a number of Hodgkin's cases that have run as long as this, but I see no evidence of Hodgkin's disease in this particular instance.

Dr. McPeak: The reason I asked was that I recently had a case in which the patient had had a repeated history of small nodules removed from the rectum. Apparently they had not been examined histologically. The one we received was a nodule of the lymphomatous type tumor, although we could not classify it because it was so small. That is why I was interested. I believe the history on that case was for ten years.

Dr. Warren: In that case I would think of two possibilities: one, the isolated polypoid growth that occurs in the rectum at times, of lymphoid origin, which is a slowly progressing process, if malignant at all; the other, a slowly growing lesion elsewhere. Another thing that has to be kept in mind there is the possibility of the lymphogranuloma. I think that sometimes when you get just a small biopsy sample, the differential from a lymphoid tumor may be troublesome indeed.

Dr. A. O. Severance, San Antonio: I would like to have Dr. Warren comment on the term "benign lymphoma," which is sometimes used in this region. Do you believe in it and do you think it is benign, or is that a misnomer?

Dr. Warren: If it is a lymphoma I think it probably is not benign. I have not seen any that I have been willing to consider as benign, with the possible exception of the polypoid lesion of the rectum, which does seem to behave differently from most of the other tumors. So far as lymph nodes are concerned, I have seen slowly growing lymphoid tumors and I have seen hyperplasias and all different types of chronic inflammations, but I have never seen anything that I felt met the criteria of a lymphoma and yet proved to be on the benign side.

Dr. Severance: I meant in just the rectum.

Dr. Warren: There is in the rectum, I believe a possibility of a lymphoid infiltration of localized character, usually polypoid, in the submucosa. I have not had enough of these myself to have any sound opinion on them. I gather from the literature that they can remain for a considerable period of time and that they can be locally removed without recurrence. I am hesitant as to how to classify them, and what I have usually done when I have gotten one of these things has been to send it to Dr. Purdy Stout and ask him what he thinks of it. (Laughter.)

Lt. Frank Vellios, Brooke General Hospital, Fort Sam Houston: You neglected to give us your diagnosis. I assume that you believe this is a lymphosarcoma?

Dr. Warren: Yes, that is my opinion.

One other point, because of the importance of the

relatively small biopsy specimen that we sometimes get and because of the frequency with which lymphogranulomatous lesions turn up, I wonder if some of you who have had fair experience with them would be willing to say something. I believe you have seen a number of these lesions, Dr. Brindley. Would you be willing to speak of the lymphogranuloma in general and how you feel as to its diagnosis by histologic means on biopsy?

Dr. Paul Brindley, Galveston: You mean the lymphopathia, the rectal stricture, and the rectal inflammation?

Dr. Warren: Yes.

Dr. Brindley: We have a great deal of difficulty diagnosing them. We diagnose them more by ruling out other things and then finding this chronic lymphoid infiltration, which is not malignant in a female. Those are about our criteria.

Dr. Warren: That was about the awkward spot I was in, and I am glad to know that I have able company there.

LYMPHOSARCOMA

CASE 12.—Contributed by Dr. D. A. Todd, Nix Hospital Laboratories, San Antonio.

Patient.—40 year old white woman (presented at the seminar by error as "4 year old white girl").

Specimen.—Scalp and cervical lymph node.

The patient reported the progressive growth for one year of a tender, painful lesion in the left temporoparietal region to a size of 4 by 2 inches. It was reddened, sometimes "hot," and occasionally throbbed. In the last seven months the lymph nodes of the left anterior cervical chain progressively enlarged. The subjacent calvarium was not obviously involved. A Wassermann test was negative. A blood count revealed leukopenia, with 3,600 white cells per cubic millimeter with 8 per cent eosinophils. At operation, no discrete tumor and no disturbance of overlying hair were found but the skin was thick and indurated. The lymph nodes were matted, hard, and gray-pink.

Dr. Warren: Our next case concerns a 4 year old youngster with the scalp and cervical lymph nodes involved. I note in this that the white count is 3,600 per cubic millimeter, with 8 per cent eosinophils. That does not mean a great deal to me because one would still have a normal eosinophil count if that is a real reduced white count. Assuming that the patient had a reduction in her polymorphonuclear leukocytes, I would not be at all surprised, with this count as low as it is, to see 8 per cent mean a perfectly normal absolute count of eosinophils. One of the things we have to remember with any low count is that it is important to weigh the absolute proportion of the various cellular elements that are present. I would consider 3.6 per cent as low. We have had many hundreds of thousands of counts made on normal persons in the course of the work of the Atomic Energy Commission and the Manhattan Engineering District preceding it. Dr. Parker and Dr. Cantril at Hanford are working up the statistics on several hundred thousands of them. One thing that has come clearly from that is that there is a much wider spread in white count than the textbooks lead one to believe. I am reluctant to consider any count above 4,000 and under 14,000 as abnormal unless there is some clear evidence in the disturbed differential count as well. I would think here that the 8 per cent eosinophils might imply that we are dealing with a true leukopenia and that some of the lobe cells or some of the lymphs have been knocked out. We do not know from the differential, since we do not have the full differential, just what happened on that.

We are dealing here with a tumor in two localities. Let us have a look at it, first from a gross standpoint.

I guess we have it microscopically in this slide instead of grossly. Here we see a tumor which is obviously infiltrating. It is situated in some fat. Here it is separating fibers of connective tissue (fig. 7). Whenever I see lymphocytes arranged in these long columns like marching soldiers, I always wonder about a tumor. You rarely see an inflammatory type of infiltration adopt those long straight lines that are fairly frequent in the tumors.

In the next slide we see a further accentuation of that line-up, and in the Masson stain we see how both the connective tissue and the fat have been infiltrated and how between the fat cells there has been built up a mass of tumor tissue and a slight amount of stroma along with it. Here we see the individual cells somewhat variable in size, recognizably lymphoid in type, with what looks like an irregular lobulated nucleus here. The reticular stain made an evenly diffused, irregular reticulum that again would go with a lymphoid tumor.

Shall we look now at the score: reticulo-endotheliosis 1, lymphocytic lymphoma 2, chronic lymphadenitis 2, reticulum cell sarcoma 1, lymphosarcoma 6, and adrenal carcinoma 1.

First, chronic lymphadenitis, I believe, can be readily ruled out. The orientation of the cells toward one another, the infiltration of the fat, the infiltration of the fibrous tissue—all are against a chronic lymphadenitis. In a chronic lymphadenitis one gets one of three things: some preservation of preexisting structure, necrosis, or scarring. I did not find that in this slide, so I am inclined to rule out chronic lymphadenitis.

For adrenal carcinoma the cells seem to me to be too small. The reticulum stain brings out an even diffusion of the reticulum fibers, irregular, delicate, which does not go with the relationship of the reticu-

lum to the clusters of epithelial cells. So, on the matter of cell size and on the matter of arrangement of reticulum, I would not like adrenal carcinoma.

The cells appear to me, in general, a bit too well differentiated for the typical reticulum cell sarcoma.

I believe I would wind up here with the lymphosarcoma. There are enough undifferentiated cells to warrant considering that we are dealing with the lymphosarcoma in this instance.

How do you feel in regard to this diagnosis? Are we all reasonably agreed on it? Are there any comments you would like to make in relation to it?

Dr. B. F. Stout, San Antonio: I would like to say first that the photomicrograph did not represent the most curious feature of these sections—the tremendous amount of fibrosis as opposed to the amount of lymphocytes in the picture. These sections were sent to Dr. A. P. Stout of New York some months ago, and he wrote back that he was uncertain as to the diagnosis.

Dr. Warren: I think that there are good opportunities for the appreciable fibrosis to occur in areas of lymphoid tumors, but when it does occur there are usually the same strands of columns of lymphoid cells left in the interstices of the fibrous tissue that we saw in this somewhat fibrotic area in this slide at the left.

Dr. Dudley Jackson, Sr., San Antonio: If Dr. Todd is here, I would like to know if that age is proper on this case. Maybe Dr. Stout knows. Is that a 4 year old patient?

Dr. B. F. Stout, San Antonio: I do not know.

Dr. Jackson: Dr. Todd was bringing out some slides on a similar case of mine, one which looks like this case. The patient was 24 years old. The original lesion was on the side of his head as best we knew. There was no hair over this area of about 3 or 4 inches wide.

Col. Elbert DeCoursey, Brooke General Hospital, Fort Sam Houston: This patient is 40 years old.

Dr. Jackson: We are still wrong on the age then.

Dr. Warren: It is interesting that there would be an added case with a clinical form so similar as yours to this. Then if you will, make the correction on case 12 that the patient is not 4 years old but actually 40 years old.

Dr. B. F. Stout, San Antonio: I would like to ask Dr. Warren if he would comment on the formation of fibrous tissue in the thyroid that will be shown along with the lymphocytic reaction; on the fibrous tissue formation in Hodgkin's granuloma; and in this present case as to whether or not the origin of this collagen is possibly from the undifferentiated mesenchymal cell that is present universally in these structures.

Dr. Warren: That is an interesting point and there is fairly good evidence that there can be a direct transition from the initial reticulin fibers formed by primitive mesenchymal cells up to the heavier and denser fibers of collagen. By and large, collagen is formed by fibroblasts and fibrocytes. The evidence that we have gained from the work of Schmitt and others interested in the details of cytochemistry would indicate that the molecular orientation in collagen is such, that the chemical nature of collagen is such... one amino acid is absent and I believe that it is tryptophane. Collagen is a fairly specialized tissue which under usual circumstances is formed by the fibrocytes.

If we take the brain, for example, where there is either an inflammatory lesion or a tumor, we find that if there is fibrosis or scarring, it can come only from the collagen-forming cells of either the meninges or the perivascular fibrocytes. If there is a reticulum cell tumor in the brain, one does not get, so far as I am aware, true collagen appearing there. I take the brain as an example simply because it is relatively free of fibrocytes. In general, I think that collagen is a product of fibrocytes, that it is formed in all probability in a suspended or in a solution state which requires the presence of ascorbic acid for the precipitation in the gel state.

One of the most fascinating things in the study of vitamin deficiencies and their repair has been the study of Wolbach and Howe. They have pointed out that in the scorbutic animal a perfectly fluid medium in what should be fibrous tissue, or adjacent to a forming tooth, or adjacent to forming bone, will be precipitated within a few hours after the administration of ascorbic acid in relatively small amounts, and that ordinarily the difficulty in the scorbutic animal is not so much the formation of the precursor of the collagen but rather the gelation, the precipitation of that collagen, the transition from the solution state into the pseudocrystalline state of the protein fibers.

The fibrous tissues in Hodgkin's disease I have always regarded as a stromal product following the foci of necrosis. I know that there is not universal agreement on this point, that there is some evidence that it may come by a combination and agglutination, a building up of the reticulin fibers, but mostly I have thought of it as the result of organization of necrotic areas.

In the case of thyroiditis, I think fibrosis rests on an inflammatory basis and comes from the proliferation of the fibrocytes and the increased activity of the fibrocytes in the thyroid stroma.

Dr. B. F. Stout, San Antonio: What I was trying to bring out was what relationship there might be

between this series of lymphatic cells and the formation of fibrous tissue, because Hatfield and Garred in their "Recent Advances in Pathology" have a scheme showing that certain ones of these cells can become fibrocytes.

Dr. Warren: I am a little worried about the evidence from tissue culture with regard to fibrocytes. One of the things that has impressed me is the difficulty of getting fibrocyte-free portions of tissue for tissue culture. Another has been the old question raised by Maximov of the origin of lymphoid cells, the relation of lymphoid cells and fibrocytic cells. I have not much of a basis for opinion, but my feeling has been that in general we do not get lymphatic cells in adult life or in postembryonic life from fibroblasts. I am a firm believer in the relative fixity of cells once differentiation has occurred. The metaplasia of cells within a given type, yes; as for example, in epithelium one can see it commonly. But I am reluctant to think of too much shifting of the fibrocytes back and forth, and then I am influenced quite a little by my study of repair in cerebral lesions where there would be ample excuse, from the standpoint of lymphoid infiltration, of macrophage infiltration, for the presence of undifferentiated cells, if they were there, to form fibrous tissue. The only time we get fibrous tissue in a reaction of the brain is when there is contact with the meninges on the one hand or the perivascular tissue coat on the other hand.

Col. Elbert DeCoursey, Brooke General Hospital, Fort Sam Houston: Some of the Spanish workers believe that they can differentiate reticulin fibers that are going to become collagen and reticulin fibers that are being made from reticulum cells. We have not been able to do that in our laboratory. May I ask how you feel about that situation?

Dr. Warren: I have not been able to do it, and I think that we either need more skill in the techniques used or that we have to wait for further detail from the study of Dr. Schmitt and his co-workers on collagen and related substances. The techniques that they have for study of the character of the protein molecule in collagen and the orientation of those protein molecules I think may carry us a long way in this point.

LYMPHOSARCOMA

CASE 13.—Contributed by Dr. J. M. Moore, Santa Rosa Hospital, San Antonio.

Patient—57 year old white man.

Specimen.—Lymph node.

The patient was struck by timber on the left side of the chest in February, 1948, with development of pain in the chest, swelling of the posterolateral left chest wall, and dyspnea. There was pleural effusion of turbid, straw-colored sterile fluid. The left lower chest region appeared dull and opaque to roentgen ray. The patient died six weeks after injury. Autopsy revealed a sheet of firm, yellow-white tissue throughout the left part of the chest wall, the diaphragm and some of the retropleural nodes on the right. An ulcerated nodule was present in the stomach, and similar tissue was seen in the left adrenal medulla and the pancreas. The left sympathetic chain appeared enlarged and was continuous with the mass in the left side of the chest wall.

Dr. Warren: The interesting thing about this case, at first, is the first statement: "The patient was struck by timber on the left side of the chest in February, 1948." He died six weeks after this. There are a number of points that we want to weigh in that. The problem of origin—are tumors caused by single trauma—plagues all pathologists from time to time. It is a great thing for the lawyers and it is a "pain in the neck" for the pathologists.

By and large, I am extraordinarily skeptical of the development of tumors on the basis of a single trauma. The best evidence that I know on this is the experience of the personnel of the Armed Forces where considerable numbers of traumas of diverse types were encountered and where there has been no undue cropping up of tumors. In the experimental animal there is little satisfactory evidence that a single trauma will precipitate a tumor; in fact, I know of no satisfactory evidence on this point. Most of the evidence rests on coincidence and most coincidences are of the sort that we have here: in giving the history the patient remembers something. Woman after woman with carcinoma of the breast remembers when she hit her breast on the corner of the table or on the ironing board or something of that sort. There are few of us, if we set our minds to it, who cannot remember trauma to some part of our body within fairly recent times. We get any number of things that do not assume importance until something occurs afterwards to fix them in our memory.

I have felt that there were certain criteria that we could use as an initial sifting in this field to simplify the problem somewhat for ourselves. In the first place, there must be satisfactory evidence that the part was intact and free from tumor before the trauma occurred. It is surprising how difficult that is to establish in most of the assumed instances where trauma is claimed for the causation of tumor. The second point is that the trauma should be adequate and should be at the point where the tumor subsequently developed. By adequate, I think we have to assume something sufficient to cause the disruption of tissue as evidenced either by a break in the skin or by a subcutaneous or deep hemorrhage.

I remember well a beautiful case of a malignant melanoma of the heel that turned up in a girl who sued the Boston Elevated Railway because she claimed that this turned up some months after her heel had

been squeezed in the closing of the car door. There was no question that her heel was squeezed in the car door; there were plenty of witnesses to that. She even had a roentgenogram made and there was a full report of the accident. The thing actually got into court before the pathologist had sufficient curiosity to see whether the trauma had occurred to the same heel that had been injured. Much to his surprise, and to the discomfiture of the claimant, the trauma had been done to the heel that was normal rather than to the heel that had developed the tumor. (Laughter.) I think that most cases of traumatic claims are about on the same basis as that.

In addition to having an adequate trauma we have to have a tumor that is of the right cell type arising there. There is not much point, if we have, let us say, trauma of the wrist and find out that the tumor there is a renal cell carcinoma, in assuming that the trauma caused the renal cell carcinoma. The time interval is important. In this case I think the time interval comes into play pretty well. We have a period of six weeks from extensive swelling of the chest, pleural effusion, to death. I think that is asking a lot even of a rapidly growing lymphoid tumor. Then we have the question of how long, from the other extreme, will we permit things to go. That is much more difficult. We know, for example, that with radiation we may have grief turning up twenty or even thirty years afterwards. However, with trauma we know that there is a much more prompt repair, that there is not a continuing injury to cells which have not been killed but only injured. I think we are fairly safe in saying that we would look with great suspicion on any tumor which turned up more than three years after initial injury, just as a rough bracket. Of course, we would look with enormous suspicion on all tumors from a traumatic standpoint anyway.

I believe all the evidence we have is much more the type of evidence that a shrewd lawyer brings to the attention of the jurist, rather than the type of evidence that the pathologist would want to consider. If those criteria are not met, until we can absolutely soundly answer the question, I think that we ought to look skeptically on the origin of tumors from trauma. If these criteria are met, there is perhaps still a possibility and it might be well to give the patient the benefit of the doubt. I think that as our knowledge advances, less and less will we see sound evidence for traumatic origin of a malignant tumor.

Now let us see what this particular one looked like. Here we have in the lymphoid type cell a diffuse infiltration through a wide area, with a suggestion that this may have been the boundary at one time of a lymphoid aggregate and an infiltration up beyond it (fig. 8).

In the next slide we have invasion of fat and invasion of a vessel. We have a characteristic lymphoid type of cell. We have a fairly even, diffusely distributed cell type, moderately well differentiated, with little mitotic activity. Here you can see it on a larger scale. The cells again look clear and reasonably differentiated.

Now let us see what the diagnostic score is: meso-endothelioma 1, neuroblastoma 1, neurogenic tumor 1, reticulum cell sarcoma 2, lymphoid sarcoma 7, and Hodgkin's disease 1. There is heavy predominance on the lymphoid type of tumor—10 to 3.

Meso-endothelioma I would not be too inclined to think of here, assuming that the usual type of meso-endothelioma originating in the pleura and reaching outward is thought of. The cells are too discrete. There is a tendency to a sheet-like formation, a compaction of the cells in meso-endothelioma that is lacking here. Also, the individual cells and nuclei are too small and too round.

Again I think that the roundness of the cells and nuclei argue against neuroblastoma.

For reticulum cell sarcoma the cells are rather too adult, although there are immature cells scattered all the way through. For those pathologists who consider the reticulum cell any lymphoid tumor in which reticular cells occur, we would have to concede the diagnosis.

I would be inclined to go along with the majority and say that this is a lymphosarcoma. I was not convinced of the presence of the Sternberg cells that I like to see in Hodgkin's disease and there is not the degree of fibrosis and pleomorphism that I like to see in Hodgkin's disease, so my choice here is a lymphosarcoma.

What are the reactions to that for a diagnosis? Anyone like to take issue with that? Any comments that you would like to make, particularly in regard to this tumor?

I think one point in the gross description is of interest here; that is that it is a yellow-white tumor. I wonder if that yellow-whiteness was due primarily to the tumor itself or to the fact that this was widely infiltrating muscle fibers. If those strands of muscle fibers with the myoglobin in them get widely separated by a lymphoid tumor, that will give a yellowish cast to the tumor that does not otherwise appear. If it was in the substance of the pectoralis, for example, I would think that the chance of the yellowish tinge being due to separated muscle fibers would be good. Otherwise, when I see a yellow or a yellow-tan color in a lymphoid tumor, I tend to think of Hodgkin's disease. I was not impressed that this was an example of Hodgkin's disease.

MACROFOLLICULAR LYMPHOMA

CASE 14.—Contributed by Dr. J. M. Moore, Santa Rosa Hospital, San Antonio.

Patient.—55 year old Negro woman.

Specimen.—Rectal mass.

The patient had had for five years a mass in the rectum which recently grew to uncomfortable size. Examination revealed a pedunculated, firm, 5 cm. mass attached to the posterior rectal wall. It was freely movable and painless and did not bleed. No lymph nodes were palpable. A serologic test for syphilis was positive.

Dr. Warren: We see in the first slide remnants of the rectal mucosa. We see lymphoid tissue with one large follicle, with another moderate-sized follicle which is elongated, with two follicles which appear to be coalescent here. This large follicle here looks as though this downward tongue of prolongation were made up by absorbing several adjacent follicles (fig. 9). Here we see well differentiated lymphoid cells in general, but a few rather poorly differentiated cells here and there.

On this next slide we see that there is relatively little, but some, infiltration of the surrounding area. With the reticulin we see that there is a diffuse, rather delicate reticulum, scanty in amount. This would fall into that group of tumors that I would like to send to somebody else to get a final diagnosis on. If I had to diagnose it on the slide as it stood, I would say that this fell within the group of the macrofollicular type of lymphomas.

Let us see what the rest of you say on this: lymphosarcoma 5, giant follicular lymphoma 5, chronic lymphadenitis 1, Hodgkin's disease 2, and reticulum cell sarcoma 1.

Those big reticulin cells I think are not Reed-Sternberg cells and there is not much fibrosis or pleomorphism, so I would lean away from Hodgkin's disease.

Lymphosarcoma is a possibility. These cells are somewhat differentiated, but they are arranged in these follicular patterns, and a lymphosarcoma does not usually take on a reticular pattern.

Chronic lymphadenitis—in the first place it is not a lymph node, and in the next place, there is not the accompanying evidence of chronic inflammation that I would like to see. I like to see some evidence of either polymorphonuclear infiltration, macrophage infiltration, some necrosis, or perhaps a little phagocytosis by macrophages before going over into the chronic inflammatory field.

For reticulum cell sarcoma I believe the cells are too well differentiated. It looks as though the overwhelming sentiment is for a lymphoid tumor, and since there are many of you willing to go along with me on a giant follicular lymphoma as want something else, let us regard that, unless serious objection turns up and unless I am convinced otherwise, as being in the macrofollicular family.

UNDIFFERENTIATED CARCINOMA

CASE 15.—Contributed by Col. Elbert DeCoursey, Brooke General Hospital, Fort Sam Houston.

Patient.—30 year old Negro man.

Specimen.—Nodule in the right breast.

Eight months prior to the present admission a hard, painful mass was noted in the right axilla. There was low grade fever, a sedimentation rate of 50 mm., and a negative Kahn reaction. The white blood cells numbered 9,400, with 8 bands, 54 filaments, 10 small lymphocytes, and 14 large monocytes. Biopsy revealed acute and chronic lymphadenitis, but there was no response to antibiotics or sulfonamide. The white blood cell count rose to 15,700, with 42 per cent segmented granulocytes. The mass was excised and reported as chronic lymphadenitis. Three weeks later a hard nodule appeared in the right breast, was excised, and again showed chronic lymphadenitis. Two weeks later another node developed in the right breast. It did not respond to roentgen-ray therapy, but did subside after therapy for lymphogranuloma (a Frei test was positive). Two months later the masses in the axilla and right breast recurred and specimens were taken for biopsy.

Dr. Warren: The significant fact is that we have a biopsy specimen which indicated acute and chronic lymphadenitis, but there was no response either to antibiotics or to sulfonamides. The other thing that interests me is that while the white blood cell count rose to 15,700, there were only 42 per cent of segmented granulocytes. Two masses were taken out and reported as chronic lymphadenitis. Then a hard nodule appeared in the right breast, which was again excised and showed lymphadenitis. Then another nodule came out. It did not respond to roentgen-ray therapy but apparently did subside after therapy for lymphogranuloma. They again recurred and were subjected to biopsy. In other words, here is a persistent lesion that keeps on recurring in spite of a wide variety of treatment, responding not at all to most.

In this slide we see first a group of cells which are arranged in a distinctly clustered pattern. I think as you look at this you will be impressed that this is a different sort of pattern from the infiltrations you have been looking at earlier. Instead of being scattered and diffuse, the cells occur in strands and in masses. Here, as we look at it, the cells are hard to make out. They are small. Some of them look like lymphoid cells. If you were as close as I am, perhaps you could see it better. The cytoplasm of the cells is more apparent and the nucleus is more sharply defined with rather more chromatin than in the lymphoid cells. Then in the reticulum, notice that the reticular fibers tend to outline little clusters of cells.

With the evidence, let us see what we have on the diagnosis: reticulum cell sarcoma 11, angio-endothelioma 1, and undifferentiated carcinoma 1.

If I were going to diagnose any lymphoid tumor I would diagnose reticulum cell because of the occurring of these definite masses. On the other hand, the masses are too clear-cut, the cells have too sharply defined cytoplasm, and too much chromatin in their nuclei for good reticulum cells. Also, the pattern of the reticulum fibers is one that more surrounds clusters of cells than simply wavers in and out indiscriminately among the cells. As a result, I think we are forced to consider that it is not a reticulum cell sarcoma in spite of the weight of the majority here.

Angio-endothelioma I would rule out because these nuclei look epithelial to me. There are normal blood vessels present and the cells are not the elongated, somewhat fusiform type that we are apt to have in the endothelial type of tumor. My own diagnosis agrees with the lone person in the minority—undifferentiated carcinoma (fig. 10).

Now the two of us will endeavor to see whether we can convince the rest of you. Who is the other one, by the way? Dr. Severance. Good work!

There is one other bit of evidence that we did not have a chance to present for you here. There was a mucicarmine stain done. I wonder if we could hear about the findings in the mucicarmine stain.

Capt. Hugo Elmendorff, Brooke General Hospital, Fort Sam Houston: The mucicarmine stain on this was negative.

Dr. Warren: Good. It is an undifferentiated carcinoma. The fact that you do not get a positive mucicarmine does not worry me.

Dr. Zola K. Cooper, Oklahoma City, Okla.: I was a little worried about the cells looking something like plasma cells.

Dr. Warren: I think that is a good point. A number of the undifferentiated breast carcinoma cells will resemble plasma cells. I think that there is this to weigh: If we had a plasma cell tumor we would have, as I mentioned earlier, a fair number of polymorphonuclear cells, which I did not find here. I think that is a characteristic of the plasma cell in general. Another thing that impressed me here was that it did not respond to roentgen-ray therapy. I think that all the plasmocytomas I have seen have responded nicely to roentgen-ray therapy. The character of the nuclei did not have a sufficiently finely defined apple-pie pattern of the chromatin to go with a plasma cell. Certainly from the standpoint of the size and shape of the cell and from the presence of rarefied areas in the cytoplasm, one would have to weigh the possibility of a plasma cell. I had rather hoped because of those rarefied areas that the mucicarmine stain was going to show something because I think that even though definite mucin droplets have not been formed, some of the epithelial cells show that sort of thing when there is a beginning secretion of mucus in it. By no means do all of the breast carcinomas secrete mucicarmine.

Col. Elbert DeCoursey, Brooke General Hospital, Fort Sam Houston: In another case similar to this, we had a female breast from which a small nodule was taken. It showed fibrous tissue with line-ups of round cells that Dr. Warren pointed out awhile ago as being in single columns like soldiers, and stated that therefore one should think of tumor. Unfortunately, we did not; we called the condition chronic mastitis. Several months later the patient came back in the hospital with a nodule in the axilla. That was taken out and it had some large round cells. That presented a terrific problem in diagnosis, some of us thinking reticulum cell sarcoma and some thinking carcinoma. A mucicarmine stain was done and in about three cells in the whole slide there were specific beautiful red dots. Although I had never gone against mucicarmine before, I did not think the condition was carcinoma in spite of the red.

We took the slide around and also got out the other tissue. It happened that Dr. Warren was through here, and we showed him this material. He immediately said that the first specimen was carcinoma and that the second was also. In the meanwhile we sent it to the staff at the Army Institute of Pathology. They came back with the idea of the thing under the arm being reticulum cell sarcoma, and said that they could not demonstrate mucin in the cells. I happened to be in New York and took the slides to show Dr. Purdy Stout. He looked at the breast and said, "Yes, this is chronic mastitis." I said, "Are you sure?" He said, "Well, anyone can see at this low power that it is chronic mastitis." I said, "Dr. Warren thinks it is carcinoma." He said, "He does! Let me look at that." Then he looked at it again and he wanted a high power view. Then he said, "Well, he's right. I should have looked under high power, but you will never see another one like this in your life." Then he looked at the axilla, at that lymphoid node, and said, "That is red all right, but that is not mucin, because mucin should be in nice globules within the cells, or else it is not mucin, even though it is red." But we were convinced that that one was also carcinoma.

HODGKIN'S DISEASE

CASE 16.—Contributed by Col. Elbert DeCoursey, Brooke General Hospital, Fort Sam Houston.

Patient.—24 year old white woman.

Specimen.—Retroperitoneal lymph node.

The patient had recurrent bouts of fever, chills, head-

aches, nausea, vomiting, and diarrhea. There was marked adenopathy of the cervical lymph nodes, with hepatomegaly and splenomegaly. She was treated with a course of four injections of nitrogen mustard (methyl bis) beginning two months before death.

Dr. Warren: You note that this case has the added interest that there has been treatment with nitrogen mustard.

In this slide we go back to our more familiar pattern of the lymphoid tumors—large masses of infiltrating lymphoid cells without the distinct architectural pattern of the normal lymph node, and with infiltration of the capsule (fig. 11).

Again in this slide we see the capsule infiltration. We see, by the way, the lymphocytes lined up like soldiers. We see here a little of the peripheral sinus still left. That sometimes happens, but we see here that it is lost and the infiltration is occurring. We see that the texture of the normal node has gone. As we look at it, we see that the nuclei are relatively few, that there is a fair amount of widely separated cytoplasm. That holds true both on this section and on the high power section as well. You will note that here the nuclei have the rather diffuse character of chromatin that we associate with the reticulum cell. On this one I have stuck out my neck by saying that it falls into the group of the reticulum cell sarcomas.

Now, let us see what the rest of you think of it. I see that I am 'way out in left field by the looks of this: Hodgkin's disease 11, pancreatic adenocarcinoma 2, and lymphoma 1.

It looks as though I will have to work hard on this one. Suppose we flash those slides back on again and let me take another look at them.

Col. Elbert DeCoursey, Brooke General Hospital, Fort Sam Houston: Did you not read your notes wrong? You wanted this slide particularly.

Dr. Warren: I had not looked at my notes; I was looking at the slides. (Looks at notes.) Yes. In this slide there is the Reed-Sternberg cell. Thanks for saving me. Yes. It shows that one should not rely too much on snap diagnosis and should look back at one's notes occasionally. I am in better company than I thought, and I will ride with the majority on Hodgkin's disease here.

With regard to the point of the carcinoma, I think the absence of distinct clumping of the cells, the absence of well defined stroma, and the absence of any alveolar formation is against an adenocarcinoma.

CHRONIC GRANULOMA

CASE 17.—Contributed by Col. Elbert DeCoursey, Brooke General Hospital, Fort Sam Houston.

Patient.—53 year old white woman, three years postmenopause.

Specimen.—Skin.

Six months before admission the patient developed a tender, wart-like growth on the skin near the rectum. A physical examination was otherwise negative and the blood picture was within normal limits.

Dr. Warren: Here we have a lesion which is in fat, to be sure, a mass of lymphoid cells. When we look at it on the higher power, we see that there is a curious pleomorphism and you note that there are eosinophilic cells. There are eosinophils scattered through the tissue here and there (fig. 12). Unfortunately, as you know, most Kodachromes do not bring out the red granules of eosinophils well, so those who are at a great distance will have to accept the eosinophils on faith; they are there.

In the next slide we have a diffuse infiltration of cells in which eosinophils, lymphocytes, and some macrophages play a major part. This shows some degree of infiltration of fat, but not the enthusiastic type of infiltration that I usually associate with tumor creeping out into fat tissue. The margin of that sort spells to me more a chronic inflammatory type of thing than a true neoplastic thing.

Let us see what the diagnoses are: mycosis fungoides 3, Hodgkin's disease 1, sarcoma 1, chronic inflammation 1, eosinophilic granuloma 5, xanthomatosis 1, and insect bite 1.

There are fat-containing cells present, but I think that is the result of phagocytosis of the invaded fat and the inflammatory process rather than a primary xanthomatous process. Mycosis fungoides and Hodgkin's disease I would rule out here on the basis of the character of the cell. There are some people who believe that eosinophils are of importance in the diagnosis of Hodgkin's disease. They are handy, they are a helpful sign, but I do not regard them as being of fundamental importance. My impression of the importance of the eosinophils in Hodgkin's disease is a good deal like the cherry in the cocktail. It is nice if it is there, it is pretty, and it is red, but the cocktail is just as good if it is not there. (Laughter.)

One interesting thing about the eosinphils is that one of the best things to attract the eosinophils is necrosis of lymphocytes. Wherever necrosis of lymphocytes occurs, and necrosis occurs commonly in Hodgkin's disease, eosinophils are apt to come in. Some of the most beautiful incidents of eosinophilic infiltration that I have ever seen have been in epidermoid carcinomas of the lip or of the cervix where there has been a peripheral lymphocytic infiltration, death of those lymphocytes, and the eosinophils coming in after them. In the healing stage of appendicitis, where there has been a necrosis of the lymphoid elements of the submucosa, we get a beautiful eosinophilic infiltration. Eosinophils alone are purely ornamental. This I do not think is a neoplastic process. I go along with those who consider it as a chronic granulomatous process. It might be an insect bite. I

do not know just how easy it would be for the average insect to get this particular region, but we will assume that it is perfectly feasible for one to do so. (Laughter.)

Any comments on this case?

Dr. Zola K. Cooper, Oklahoma City: How do you rule out eosinophilic granuloma?

Dr. Warren: I cannot. That is a granulomatous process. However, the fact that I am interpreting these cells containing fat as probably a local phagocytic thing, rather than as part of a lymphoid disturbance, would swing against an eosinophilic granuloma as such.

Dr. B. F. Stout, San Antonio: I might say first that Fred Weidman of Philadelphia has written extensively on eosinophilic granuloma of skin, and he is trying to connect it with eosinophilic granuloma of bone. I understand that both conditions have been found in the same patient. I am wondering now just what your observations would be on that.

Dr. Warren: I have not had much experience in this. The eosinophilic granulomas of bone that I have seen have happened to be without skin lesions. Is there any lesion of bone in this patient? That might have an important bearing.

Dr. Stout: So far as I know—no.

Col. Elbert DeCoursey, Brooke General Hospital, Fort Sam Houston: There is none.

Dr. Warren: I would have to leave that open. I would be willing to accept here a diagnosis of a granulomatous process in which eosinophils predominate without trying to run it down too much beyond that point.

Dr. John H. Liles, Jacksonville: Is it true that in lymphosarcoma one can foresee an infiltration of the fat more than is seen in Hodgkin's disease?

Dr. Warren: I do not believe I could say on that. I have seen about the same degree of infiltration of the fat at the periphery of Hodgkin's lesions as I have of lymphosarcomas. I think it hinges somewhat on the rate of growth of the Hodgkin's. If you have a slowly growing Hodgkin's process, you will not have as extensive or obvious an infiltration of adjacent fat. There are many forms of Hodgkin's which are exceedingly slow growing. I do not subscribe to the fine subdivisions of Hodgkin's disease that Dr. Jackson and Dr. Parker of Boston do. I am inclined to think of it as probably a single disease process with varying phases, and I have found great difficulty in distinguishing the granuloma, the perigranuloma, the sarcoma, and so forth. By and large, I have made a histologic diagnosis of Hodgkin's disease on the histologic findings and then have taken into consideration with the clinician the character of the lesion, the extent of the lesion, and the time involved, and tried to guess on that as to whether it is a slowly or rapidly progressing thing. The histologic criteria that have been suggested in the articles of Parker and Jackson I have not been able to apply successfully.

HODGKIN'S DISEASE

CASE 18.—Contributed by Col. Elbert DeCoursey, Brooke General Hospital, Fort Sam Houston.

Patient.—25 year old white man.

Specimen.—Small intestine.

An abdominal mass the size of a golf ball first appeared three months before admission. During the ensuing weeks the mass reached about 8 cm. in diameter. The patient lost 10 or 15 pounds and looked chronically ill. The mass was in the epigastrium and was not attached to the stomach. Study by roentgen ray of the upper and lower portions of the gastrointestinal tract were negative, and retrograde and intravenous pyelograms were negative. A chest roentgenogram was negative. The urine was normal. The patient had a daily irregular fever of from 100 to 101 F. The small intestine was grossly thickened, red-gray, and firm in focal areas.

Dr. Warren: In this slide we have a spleen that is appreciably involved, a spleen which on section has mottled, pale areas in the pulp which are not too clearly suggested, although there is a suggestion of them down here in the capsule. There is no definite thickening of the capsule. The shape is maintained and the splenic notch is reasonably well present still, in spite of its increased size.

Here we see in the vertebra a nice example of a pale, infiltrating lesion which is replacing the marrow and has replaced to some extent the vertebra itself. A significant thing here is that it is not restricted to the marrow cavity but extends diffusely up to and into the intervertebral disk and the fibrous tissues.

In the next slide we see the lesion in the gastrointestinal tract, and here it is again localized in the small intestine. That gives us an excellent gross picture of this widely diffused lesion.

Microscopically, we have here a section from the intestine, where the mucosa is again lost and there is no polymorphonuclear infiltration. There is a diffuse cellular infiltration without distinct stroma, with separation of the preexisting supporting structures and a number of large cells scattered through.

In this slide we see that those cells are predominantly of lymphoid type; that they are no respecters of boundaries, that they have crept through and distorted the normal tissue patterns, and that there are large cells of the Reed-Sternberg type present. There is pleomorphism present (fig. 13). Incidentally, I do not see any eosinophils in this particular area. Here is a fairly heavy laying down of reticular fibers in an irregular pattern. Here again there is a diffuse fairly extensive fibrosis with a pleomorphic type of infiltration. Here is the mucosa with persisting glands

still present, even though the large abnormal cells of lymphoid or of Reed-Sternberg type are creeping out among the mucosal glands. Here again is a diffuse reticular pattern which is not characteristic of epithelial cells but more characteristic of the lymphoid tumors. Farther over here are large cells, some of the Reed-Sternberg type, and some with the smaller immature lymphoid cells, or even mature lymphoid cells, scattered in among them.

I think we ought to get good agreement on this. Let us see what the score is: Hodgkin's disease 5, lymphosarcoma 2, clasmatocytic lymphoma 1, reticulum cell sarcoma 2, and carcinoma simplex 1.

Carcinoma simplex I would rule out on the basis of the reticular pattern, of the diffuse character of the infiltration, of the persistence of the intestinal mucosa, of the fact that it metastasized to the mucosa of the intestine—that is unlikely for a carcinoma; its metastasis to the spleen is unlikely also—and of the fact that there was infiltration of the intervertebral disk, which practically never happens in a metastatic carcinoma. The intervertebral disk will maintain itself intact.

Reticulum cell sarcoma I would consider. There are many immature cells of reticular type, but there are also the Reed-Sternberg cells.

Clasmatocytic lymphoma I think is almost synonymous with Hodgkin's disease, and I think we can settle on Hodgkin's disease as the essential diagnosis.

Any questions or discussion on this tumor?

I would like to bring out that this case again illustrates the fact that when you get a small intestinal tumor that is metastatic, you should think of a lymphoid tumor, a malignant melanoma, or a leukemic infiltration. Carcinoma is rare there. Even carcinomas of the breast, when they spread more widely, far more frequently involve the peritoneal surface of the intestine and grow through, pushing the normal mucosa ahead of them, even when they are right up against it, than develop in the mucosa of the intestine.

Another point to keep in mind is that metastatic carcinoma, when it goes to bone, is slow to infiltrate structures adjacent to bone. It stays put where it lights. When you find a tumor that involves both the substance of the vertebra and the substance of the supporting connective tissue, as well as of the intervertebral disk, think seriously of a tumor of the lymphoid type. You will be right nine times out of ten if, just on that alone, you say you are dealing with a lymphoid tumor.

Any questions on this particular tumor? I am pleased to see that as we go along we are achieving more and more agreement. At least, we are in the same boat, whether it is going in the right direction or not. (Laughter.)

The Pel-Ebstein fever is worth a brief word. I have been disappointed in many of the recent Hodgkin's cases as to how infrequent the Pel-Ebstein becomes. I think its presence primarily implies that there is necrosis occurring in some portions of the tumor. I think that it is not any special feature of the Hodgkin's process as such, but related to the occurrence of necrosis in the disease process. I have seen metastatic carcinoma of the breast and I have seen other types of carcinomatous lesions in which you get just as nice a Pel-Ebstein type of fever when necrosis and breakdown of the tumor is occurring.

Col. Elbert DeCoursey, Brooke General Hospital, Fort Sam Houston: Do you ever use the term "Hodgkin's sarcoma"?

Dr. Warren: I sometimes do, or I am more apt to use perhaps the not too good term of "cellular Hodgkin's disease" when there are a great number of the Reed-Sternberg cells. I would say that this certainly fell in the type that is ordinarily classified as Hodgkin's sarcoma. I do prefer to think of it as essentially a single disease process which may be granulomatous in general character but which is none the less potentially neoplastic.

I might say in passing that if we are able to set off any group of tumors from the true neoplasms at one time or another, I think we are apt to wind up by putting the lymphoid tumors and the leukemias in the group that are caused possibly by virus or by some other agent that may be well recognized. There is a good bit of evidence in the case of Hodgkin's disease, for example, that there may be some definite contributory factors. Some of you have read Grand's work, and you know Hoster's interest in the apparent appearance of inclusion bodies in the Reed-Sternberg cells. I am not convinced that they are true inclusion bodies. I am not convinced of the viral origin of Hodgkin's disease. I feel that so far we have to consider it as essentially a neoplastic lesion of unknown etiology.

It is worth while, since brucellosis was spoken of this morning, to put in just a passing word on the assumed origin of Hodgkin's disease on the basis of brucellosis. I think that Dr. Forbus has now come to the conclusion that the cases he thought were significant from this standpoint were simply due to the high level of endemic brucellosis in the population as it existed in the North Carolina area. I think from my last conversation with him that he no longer feels that there is any significant relationship between brucellosis and Hodgkin's disease. There has been some evidence from time to time in the Gordon test, in the diagnosis of Hodgkin's disease, and there has been some thought that this threw evidence on its

viral nature. Have any of you been interested in the Gordon test? It is essentially the intravenous inoculation of a mouse—I believe a ferret was the original experimental animal—and the development of an encephalitis that is apparently a nonspecific response, is not consistent, and is of no real value in the final diagnosis of Hodgkin's disease. I think our diagnosis has to rest on histologic evidence rather than on any other evidence at present. I do not feel that there is any evidence bearing on the infectious origin of the process.

Dr. M. Gerundo, Lubbock: What do you think about some work done by Frank Carter on the integral form of tubercle bacillus?

Dr. Warren: There has been a great interest in the association of Hodgkin's disease and tuberculosis. In fact, those of you who went to Vienna before the war will remember the popular pathologic lecturer there, Erdheim. Primarily as a stunt, I think, he would put on rubber gloves and wear them when he was doing an autopsy in a case of Hodgkin's disease. He felt the association with Hodgkin's disease in tuberculosis was significant. We have tried any number of animal inoculations, and as in the case of sarcoid, they have been without success. As you know, Dr. L'Esperance once felt that avian tuberculosis was of importance in the etiology. There is no question but that there is an association of tuberculosis and Hodgkin's disease. I do not think that it is more than a chance association. It is significant that through pasteurization of milk there has been less lymph node tuberculosis, and we have seen significantly less coexistence of the two. In England, where they still revel in freedom and in tuberculosis which is milk-borne because they do not want to pasteurize their milk, they are still getting a higher degree of association than we are getting. I think that probably it is a chance association rather than an etiologic association.

Dr. Gerundo: I cannot remember now the names of two French authors who recovered the virus in the second generation and finally secured the culture of the bacillus in the third generation.

Dr. Warren: I think that if we were dealing with a process where the protein of the tubercle bacillus were present, either in filtrable or nonfiltrable form, we should have positive evidence from the tuberculin test. Because of the inconstancy of the tuberculin response, I have been reluctant to accept this hypothesis. I think there is a fair chance of coexistent infections and perhaps an overlap in this way, but I have not felt that there is enough evidence to overcome the strong negative evidence as to the real etiologic significance in this case.

RENAL CELL CARCINOMA, METASTATIC

CASE 19.—Contributed by Col. Elbert DeCoursey, Brooke General Hospital, Fort Sam Houston.

Patient.—54 year old white man.

Specimen.—Biopsy of a mass in the sixth left rib.

For thirteen years there were successive painful bony lesions of the right acetabulum (1934), lumbar spine (1940), left hip (1941), and right shoulder (1941), with relatively symptom free intervals following intensive roentgen-ray therapy to the lesions. Fractures of the right humerus (1943 and 1947) and left clavicle (1947) occurred after negligible trauma, the humeral fracture upon the latest admission (1947) being ununited but painless and the clavicle quite painful. Examination also revealed painful masses in the left sixth rib and left humerus. By roentgen ray the recent lesions showed extensive rarefaction and swelling of the bone shafts; the old lesions were sclerotic. Laboratory data included the following: Kahn reaction negative; urinalysis normal; blood, moderate normocytic anemia, with normal white blood cell count and differential, sedimentation rate 52 mm., blood calcium 11 mg. per 100 cc. phosphorus 4.5 mg. per 100 cc., acid phosphatase negative, 17-ketosteroid 18.2 mg. excreted per twenty-four hours as androsterone, cholesterol 160 mg. per 100 cc. (esters 67.2 mg.), and total blood lipids 710 mg. per 100 cc.

Dr. Warren: This slide is the pretreatment lesion, as I recall it, and we notice here a distortion and a rarefaction occurring in this region.

In this posttreatment slide is a distinct ossification of the area previously involved. Now, in this, I am inclined to think that we are dealing in part with a response to the disappearance of the tumor and in part, perhaps, to some degree of radiation osteitis because of the great density of the bone. Are there any of you who would care to comment from the roentgen-ray standpoint on these two films? If not, we will go on to this film which brings out the occurrence of the lesion that developed in the rib. I believe it is from this one that your slide came.

This next is a spot film. Here is the margin of the heart and pericardium. Here you note the course of the ribs, catching the anterior ribs as they come down, and here is the mass of the tumor.

Finally, over here is a picture after roentgen-ray therapy. There has been a pathologic fracture here. There has been a distinct increase in density of the humerus.

Here you see the tumor from the rib itself. Here is the rib and there is extensive yellow swelling with hemorrhagic mottling.

Here is a section of the primary tumor itself. You can see a portion of the kidney remaining, with the ureter down here. This gives a premonitory suggestion that we are dealing with a renal cell carcinoma —yellow, translucent, grayish-green edematous areas, hemorrhagic areas. This is practically pathognomonic as we look at it in gross.

Then when we look at it from the microscopic

standpoint (fig. 14), we see that in the bones there are these curious isolated cells, large and small, with destruction of bone, with loss of endosteal activity along the bone, and a great deal wider separation of the bony trabeculae than is normal. Some of these cells are large, and from this alone one would have great trouble in making the diagnosis.

Let us consider how troublesome that diagnosis really was. I thought it would interest you to read some of the opinions that were given when this tumor was sent out from the Army Institute of Pathology. Dr. Fred Stewart said, "This case corresponds to what I thought represented primary liposarcoma in bone in 1931 and 1932. Since then I have given up this diagnosis, for there is no such disease. I do not know what these cases are. We have a number in our files but we are unable to classify them." Dr. Purdy Stout said, "Lipoidal histiocytosis, probably Gaucher's disease." Dr. Henry Jaffe said, "This may be the adult counterpart of Letterer-Siwe's disease or a progressive, malignant form of Schuller-Christian's disease." Dr. Sophie Spitz said, "Metastatic carcinoma, possibly of renal origin." Dr. Granville Bennett said, "A typical myeloma, possibly lipoblastic myeloma." Dr. Sidney Farber said, "Not a lipoid storage disease of any sort; tumor, possibly liposarcoma." You pay your money and you take your choice! (Laughter.)

I think at this stage it would be interesting to leave ourselves in suspense while we look at the rest of the slides.

In this slide we see a larger magnification which begins to look a little more reasonable. Let us leave this for the time being. That sort of gives the story away.

Here we see these cells very large. They rather give it away too.

But look at them in this slide where they are puzzling and queer, and here where the reticular net is not too helpful. Owing to the fact that there are round clusters of cells, I would think of carcinoma.

Again, in the clustering phase here, I think of carcinoma—large cells, sometimes containing fat droplets, with well defined nuclei, with definitely demarcated chromatin, sharply outlined nuclear membranes, clear-cut polyhedral cells that make a pattern with one another, large in size.

Finally, when we look over here, we come back to the same conclusion that we got from the gross. This cannot be anything except renal cell carcinoma. I would make a flat-footed diagnosis from the gross alone, or from this slide alone, of renal cell carcinoma.

Let us see what the score was from your standpoint: xanthomatosis 1, lipoid histiocytosis 3, reticulo-endothelioma 1, undifferentiated carcinoma 1, lipoid storage disease 1, endothelial neoplasm 1, and carcinoma, metastatic 4.

Just as I thought. You are having the same grief that all the rest of us had. One was pretty close with an undifferentiated carcinoma. Four hit the jackpot with metastatic carcinoma.

This is an extraordinarily interesting tumor in the way that it is spread out. I think that anyone would have to say on the basis of the initial slide that the diagnosis could not be accurately made. The slide which we had came from the biopsy specimen of the lip and is a little clearer than the initial slide, which, as I remember it, was shipped around and which came from the pelvis.

Lt. Col. Alfred Ackerman, Brooke General Hospital, Fort Sam Houston: I would like to make only one correction: The humerus which you saw was not treated with radiation, and the density of the bone was not the response to radiation therapy. That was a reaction which occurred spontaneously over a period of years. That would make it particularly interesting because the lesion was always limited to the bone and apparently never extended beyond the periosteum.

Dr. Warren: That is extraordinarily interesting. I am grateful for that comment because it would be of some interest if it had been as I thought, a radiation osteitis, but the fact that we have gotten this type of response in a slowly progressing lesion is of extraordinary interest. I think we are all too prone to think of renal cell carcinoma as always producing a bone destruction type of lesion, an osteolytic form of lesion.

Lt. Col. Ackerman: I think the second thing of interest is that the sclerosis which you saw in the iliac bone following radiation was only a temporary feature in the course of this patient's disease. Toward the end of his hospital course, as a matter of fact, in 1946, he had a complete restoration of his normal bone pattern in this particular area, so that in some areas the regeneration went on through the various stages to complete reorganization and a normal bone trabeculation.

Dr. Warren: That is interesting indeed. You certainly have picked out a beautiful case from this standpoint, not only from the diagnostic problem, but from these changes in the bone that have subsequently occurred.

Lt. Col. Ackerman: The third thing is that I felt rather late that it was a renal carcinoma simply because over a period of thirteen years of recurring observation in the hospital the patient had no urinary symptoms and no one ever found anything wrong with his kidneys. For that reason they were not examined. When we began to suspect that there was

calcification in the region of the kidney, which was not present originally, we were able to demonstrate a defect with an intravenous urogram. In spite of the pathologic diagnosis of adrenal cortical tumor, I thought that renal cell carcinoma was more likely than adrenal carcinoma. Do you think it is renal cell carcinoma?

Dr. Warren: I think that it looks more like renal cell than adrenal cell. There is, of course, always the problem of what the so-called hypernephroma is, but I think evidence has accumulated that this is more of the renal cell type than of the adrenal cell type. I would like some of you who are better clinical pathologists than I to comment on the level of the 17-ketosteroids here. Do you think that there is a significant elevation of them in this case? I would say that it was of normal level. If this were an adrenal cell tumor I would expect some elevation of the 17-ketosteroids.

Dr. C. T. Ashworth, Fort Worth: Is there any possibility, from the histologic appearance, of this being periganglioma or pheochromocytoma? I would also like to ask if the gross examination of the kidney made it clear that the tumor originated in the kidney rather than in the suprarenal gland?

Dr. Warren: Perhaps I can pass that last question to Colonel DeCoursey. Was it clearly of renal origin at autopsy?

Col. Elbert DeCoursey, Brooke General Hospital, Fort Sam Houston: Yes.

Major Milward W. Bayliss, Brooke General Hospital, Fort Sam Houston: At autopsy, as near as I could tell, both kidneys were in the usual condition that one would expect. Then there was this large tumor mass, as you saw it in the Kodachrome, sitting on top of the kidney.

Dr. Warren: Actually then, this was outside the capsule of the kidney. I would still, in spite of that, think more of renal than adrenal origin, because of the fact that in the development of the kidney there are embryonic remnants of the primitive renal precursor cells that may remain scattered outside.

This did not look to me like a typical pheochromocytoma, although they are difficult to recognize. There is nothing in the history to suggest disturbance of the blood pressure, and I am almost sure that over all the years, if this were a pheochromocytoma, there would have been some evidence of that. Sometimes it is hard on individual sections to differentiate the cells of one from another. I have been wrong and have been straightened out by the hormonal studies only. I will not guarantee it, but I would prefer to think, from the standpoint of the clinical behavior, the available evidence here, the natural history of the renal cell tumor as against the pheochromocytoma, and the gross appearance, that we were concerned more with a renal cell tumor than with an adrenal tumor. I am sticking my neck way out on that, I realize.

Dr. Paul Brindley, Galveston: I would like to have you discuss the question that comes up sometimes of trying to remove a solitary metastasis from one of these tumors in the lung.

Dr. Warren: In a renal cell tumor I think there is more justification for the removal of a solitary metastasis than for probably any other. I have seen several instances where it has been attempted and carried out successfully. There is no tumor that is more apt to metastasize more bizarrely than this, and to permit the patient to live longer in the presence of metastases than this. I am rather inclined to think that there is a reasonable chance, if you are sure that there is only one metastasis and that you have gotten the primary tumor out, if you go after the local lesion even though it be in the lung. I recall one instance where the metastasis was in the lower end of the radius. A resection of the lower end of the radius, together with a resection of the primary tumor as well, resulted in the patient staying well for four or five years, after which I lost track of him. The tendency to solitary metastasis, well demarcated, or perhaps multiple, occurring at long intervals, is great in this renal cell group.

Dr. Leo Lowbeer, Tulsa, Okla.: One case is reported in which after the removal of a renal cell carcinoma (removal of the kidney), several years later a single metastasis was noticed by radiography in the lung. After a lobectomy the patient survived five more years.

I would like to ask whether on autopsy in this case a tumor from the renal or suprarenal vein or the inferior vena cava was found?

Major Milward W. Bayliss, Brooke General Hospital, Fort Sam Houston: No, sir.

Dr. Warren: The invasion of the renal vein is frequent. Of course, here we have an extrarenal lesion, so I would look in the retroperitoneal vessels rather than in the renal drainage area itself. I think the fact that we have metastases distributed, as these are, to bone practically forces us to assume that it is a hematogenous spread of the tumor.

HODGKIN'S DISEASE

CASE 20.—Contributed by Dr. Warren.

Patient.—32 year old man.

Specimen.—Skin from the leg.

The specimen consists of a rounded 0.7 by 1 cm. nodule of tissue with a pink, smooth surface. On the cut surface the tissue is pale and homogeneous with a 0.1 cm. smooth capsule.

Dr. Warren: In this slide we have a mass of lymphoid cells, ill-defined and infiltrating out into the adjacent tissue in the corium where they have no business to be.

Here we see that they are well differentiated cells, that they are diffusely distributed, and that they are separating the normal fibrous tissue.

Next we see what looks from here like a Sternberg cell, and there is some pleomorphism of the cells. The reticular pattern is that which would go with a lymphoid tumor (fig. 15).

Let us see what the diagnoses are: histiocytoma 2, lymphocytic leukemia 2, lymphatic nodule 1, infectious granuloma 1, Hodgkin's disease 3, leukemia cutis 1, mycosis fungoides 1, lymphosarcoma 1, and chronic inflammation 1.

I am going to decide with the group that favors the neoplasia, rather than chronic inflammation, and I am going to side with those who believe that this is in the Hodgkin's group, because it looked like there were good Reed-Sternberg cells present.

If it were lymphatic leukemia, I would expect to see a more diffuse infiltration which would be somewhat perivascular in distribution. Frequently they are a good deal closer to the skin, more superficial in the corium, than this nodule is, although that is not a constant finding.

Dr. William W. Tribby, Memphis, Tenn.: I was not convinced about the Reed-Sternberg cells and I thought that there were lymphoid nodules with germinal centers there, which to me would be rather unusual for Hodgkin's disease. I would like to know how you feel about that.

Dr. Warren: I am not convinced of there being good germinal centers. If there were, I would agree with you. That would be pretty much against Hodgkin's disease. But I am not sold on there being germinal centers. I think it is more an accumulation of lymphoid cells. There are enough cells of irregular contour and of irregular nuclei to make me think it could be put over into the Hodgkin's group.

PAPILLARY CYSTADENOMA

CASE 21.—Contributed by Dr. Warren.

Patient.—36 year old woman.

Specimen.—Thyroid gland.

The specimen consists of a single lobe of thyroid tissue weighing 15 Gm. and partially covered by previously incised capsule. The majority of the lobe consists of beefy red thyroid tissue, while one pole is occupied by a roughly ovoid, well-demarcated, partially calcified, hard yellow nodule with a vague interior lobulation. Immediately adjacent is a well-demarcated, previously incised, empty cyst with hemorrhagic lining and a wall which in some areas is opaque and yellow, and measures up to 0.3 cm. in thickness.

Dr. Warren: Here I am getting back to my special favorites again.

In the first slide we see first a distinctly papillary type of tumor. In huge nodules of colloid goiter you sometimes get bizarre papillary projections, but you never get them looking like this. They are always little clumps of thyroid follicles rather than these long aberrant strands, which usually are covered with a columnar type of epithelium. I think you can get the high cuboidal or the columnar type shown fairly well here. This, I would say, from its structure, is a papillary cystadenoma. Here and there are diffuse areas of fibrosis and scarring where presumably there has been hemorrhage into a cyst, and fibrosis, and even late persisting of small numbers of the tumor cells. In this particular tumor there was blood vessel invasion. The sections that we have, unfortunately, do not show that; at least I did not find it, and I do not think that others found it. Consequently, on the basis of an infiltrating lesion which does not stay put, which has some evidence of blood vessel invasion, which persists in the face of scarring, and which is distinctly neoplastic, we would toss this in the group of papillary cystadenomas of the malignant type.

Now let us see what the diagnoses are: thyroid, papillary cystadenoma 9, thyroid, papillary carcinoma 2, thyroid, Riedel's struma 1, and thyroid, Hürthle cell tumor 1.

Good. Nine agree, and two practically agree—just a minor difference in terminology. It is not Riedel's struma because the papillary projections do not occur in that disease. The fibrosis that is present could go with Riedel's lesion, but Riedel's would not have the particular type of epithelium that persists in the midst of fibrosis. It would have poorly formed thyroid follicles and the cells probably would be Hürthle cells. I do not agree with the Hürthle cell tumor because, while these cells are tall and columnar, they do not have the glossy, eosinophilic character that goes with the Hürthle cell.

I thought it might interest you to check over a little the age incidence of this particular series. You note that this woman is 36. In a group of these that Dr. Feldman worked over in my laboratory he found that a great majority occurred before 30 years of age; that out of a group of these tumors 49 occurred before the common prevalent tumor age of 50; and that only 8 occurred after 50. When I was in Dr. Maxfield's clinic in Dallas yesterday morning, I saw a little girl of 4 who had this type of lesion, and in our series there were several youngsters. The age group that had more than any other was the twenty-first to the thirtieth year, with 20 cases in our series.

Another point of interest is whether these are of thyroid origin or of lateral aberrant origin only. In the group that we had, we had a total of 57 where

there was satisfactory evidence as to origin. Of these, 49 apparently originated in the thyroid, and only 8 were apparently present in lateral masses alone. Finally, of the group, there were only 11 which failed to show either blood vessel or lymphatic invasion. We are gradually being forced to the conclusion that most of the so-called papillary cystadenomas of aberrant thyroid origin do not originate from aberrant tissue but are actually metastases from the thyroid itself. There are a few instances in which they are definitely aberrant tumors, but they are much fewer than the occult. If one examines them carefully, there is practically always some evidence present in the course of the substance of the thyroid. This means that if we operate upon a case of lateral aberrant thyroid and take out only the lateral tissue, we are practically guaranteeing that the patient will not be cured. We are taking a strong chance of leaving tumor tissue behind in the thyroid. It is my feeling that it is best in operating upon a lateral aberrant tumor, if it is unilateral, to take out that lobe of the thyroid and the isthmus as well. If it is bilateral, as they all too frequently are, a total thyroidectomy should be considered. These cases, fortunately, are singularly responsive to roentgen ray, so it is well worth while, even if they look inoperable. I have seen these lesions when they were fungating through the skin of the neck and emergency tracheotomies had to be done to keep the patient breathing. It still pays to treat them with roentgen ray, and sometimes you can get, if not cure, so much shrinkage that you can go at them from the standpoint of radical surgery and salvage what looks like an absolutely hopeless case.

LEUKEMIA

CASE 22.—Contributed by Dr. Warren.

Patient.—57 year old woman.

Specimen.—Kidney.

The history began with a nervous breakdown and emotional instability which gradually cleared in one year, nine years prior to the present admission on June 19, 1937. Eighteen months before this admission the patient tired easily and was out of breath, but symptoms were otherwise negative. A fibroid of the uterus was noted, and treated six months before with four roentgen-ray treatments. The patient's "run-down" feeling gradually became worse. Two months before she had mild hot flashes and some nausea, but no loss of weight. Especially on exertion there were occipital and postaural constant headaches. She noted tingling and a feeling of going to sleep for three months. For three weeks she noted a poor color.

Physical examination on admission revealed the following: tongue negative, irregular mass in the uterus, external hemorrhoids, reflexes normal, soft blowing systolic murmur of the heart, and slight tenderness in the right epigastrium. There was a suggestion of a lemon yellow color of the skin and pale mucous membrane. On June 21 the red blood count was 2,250,000; hemoglobin 59 per cent; polymorphonuclear leukocytes 60 per cent; lymphocytes 32 per cent; monocytes 4 per cent; and megaloblasts 4 per cent. Twelve cells could not be classified. Platelets were decreased and a Hinton test was negative. On June 22 liver therapy was started. In a count of 100 white blood cells there were 45 nucleated red blood cells and megaloblasts. There was anisocytosis, poikilocytosis, and stippling. There was no subjective improvement with liver therapy. On June 24 the red blood cells numbered 2,050,000; hemoglobin was 45 per cent; and the color index 1.1 Gastric analysis showed 15 per cent total acid. The reticulocyte count varied from 2.8 to 4.6 per cent. On June 28 the red blood cell count was 1,840,000; white blood cell count 16,800; and hemoglobin 42 per cent. The differential count showed 15 per cent megaloblasts, 20 per cent normoblasts, 21 per cent polymorphonuclear leukocytes, 30 per cent lymphocytes, 3 per cent eosinophilic leukocytes, 6 per cent monocytes, and the remainder unclassified. Twelve reticulated red blood cells were seen. On July 7 there were 16 per cent polymorphonuclear leukocytes, 45 per cent lymphocytes, and 39 per cent unclassified, apparently of blastic origin. On July 14 the red blood cell count was 1,480,000; white blood cell count 44,300; and hemoglobin 30 per cent. The patient's jaws and gums were tender, but there was no throat ulceration or hemorrhages. There was some nausea and vomiting. Roentgenograms of the body and bones were negative. The patient's course was downhill, and she died July 15, 1937.

Dr. Warren: Now we come to our last case.

Here in the kidney you see irregular infiltrations of tumor cells here and there, interstitial infiltration of tumor cells (fig. 16). Under higher power you can see them more clearly. Under still higher power you can make out a little of their cell type. Here you see one of the casts that formed in this case, and there were others of those casts.

I would like to call your attention to the blood findings in this case. You will note that the patient has a persistently developing type of anemia, that she has a high proportion of nucleated red cells, and that finally she turns up with a large number of unclassified cells, apparently originating from the immature series (39 per cent unclassified immature cells).

I think if we can get the collecting tubule area of this kidney we will see a little more of the casts than we have seen on these other slides. It is not so good in this next slide either. I am afraid that my memory of these slides is not as good as it should be. I had remembered more casts being present. Here and there are a few.

Let us see what the diagnoses are: leukemic monocytoma 1, leukemic infiltration 3, leukemia, monocytic 1, leukemia, myelogenous 5, and erythro-leukemia 1.

We are all agreed that we have some queer type of leukemic process. I am afraid I am going to leave you on a note of some degree of uncertainty in this particular case. I would be glad for you to puzzle over it and to send me your comments if you come to any conclusion as to just what sort of leukemic process we are dealing with here. It is evidently an

undifferentiated form. Whether it comes from one of the primitive elements, the myeloid side, from poorly recognizable plasma cell leukemia, or from some of the undifferentiated lymphoid leukemias, I find it hard to make up my mind, and I would be misleading you if I tried to put down any flat diagnosis in this group.

Dr. B. F. Stout, San Antonio: I am wondering if this blood picture that is shown might not be compatible with that group of diseases described as leuko-erythroblastosis or myelophthisic anemia?

Dr. Warren: I think that would be a distinct possibility. I have not been able to satisfy myself as to just which pigeonhole it goes into. I think that is a reasonable one to weigh because you will notice in the history this tremendous proportion of nucleated red blood cells that has been spoken of all the way through and which might constitute a significant factor.

Dr. Stout: Have you ever seen a case of leukemia arising from the red cell precursors alone?

Dr. Warren: I have not recognized it if I have. I am not at all sure but that polycythemia vera may not be an equivalent of leukemia, involving the precursor of the red cell series, but the evidence is not sound on that, and I have not seen anything that I could recognize as a true erythroblastic type of neoplastic proliferation and erythroblastic bone. Of course, many of the polycythemias will ultimately wind up with a leukemic picture.

Dr. Stout: Were the bones examined by roentgen ray or was the bone marrow examined in this case?

Dr. Warren: The bones were not subjected to roentgen ray. As I recall it, the patient complained of pain in the bone, which would go with the probability of some sort of proliferation in the marrow. I do not recall what we found in the bone marrow. I did not bring my notes on that, but I am pretty sure that there was a distinct abnormality of the bone marrow.

Dr. Stout, will you take over now, please. I think that completes our run of cases. (Audience stood and applauded.)

Dr. Stout: I am sure that all of you have been rewarded for this day by the instruction that Dr. Warren has brought us. We are all exceedingly grateful to him for taking this day from his very busy life to come down here to speak to us. He said he would like to get back into pathology again, and he has gotten back into it in a big way.

I want to thank once more, or to back up the thanks that Colonel DeCoursey gave Major Stansell. I am sure that all of you have been terrifically impressed, as I have been, with the amount of work, and the extra work, which he has done in preparing these lantern slides and these photomicrographs, and with the scheme that has been used of having four screens in order to save time and to allow comparison, which is something unique and which we will adopt again next year. I hope you will all be back next year and that you will bring your friends with you. Let us stand and give a hand to Colonel DeCoursey and his officers for this wonderful reception. (Audience stood and applauded.) The meeting is now adjourned.

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ROENTGEN-RAY DIAGNOSIS OF Q FEVER

Roentgen rays can play an important part in the diagnosis of Q fever, report three Los Angeles physicians in the November issue of *Radiology*. Drs. George Jacobson, Ross B. Denlinger, and Ray A. Carter state that chest films of Q fever patients show to what extent pneumonia has developed and that roentgen rays reveal the exact pulmonary involvement. The physicians believe that Q fever is often confused with primary atypical and virus pneumonias, influenza, and meningitis and is overlooked.

None of the new drugs has had any demonstrable effect upon the duration or severity of the disease; however, aureomycin was tried in only 1 case with apparently favorable results. Patients usually recover from the disease and one attack confers immunity for an indefinite period.

The first known outbreak of Q fever occurred in Amarillo in March, 1946, with 55 cases, including 2 deaths. More than 300 cases have been recognized in southern California since May, 1947. The incidence is highest among meat packers, laboratory workers, and persons working in or living near dairies.

APPROVAL OF SULFATHIAZOLE WITHDRAWN

The American Medical Association Council on Pharmacy and Chemistry has withdrawn its acceptance of sulfathiazole and sulfathiazole sodium, according to the September 24 *Journal of the American Medical Association*.

"The Council recently considered the present status of sulfathiazole," the statement says. "It considered the fact that approximately 18 per cent of patients who receive sulfathiazole develop untoward reactions such as fever, rash, acute leukemia, leukopenia, and other manifestations of toxicity (which compares with about 16 per cent for sulfapyridine, 12 per cent for sulfanilamide, 6 per cent for sulfadiazine, and 7 per cent for sulfamerazine).

"Further question of the need for continuing acceptance of sulfathiazole was raised in view of the fact that less toxic sulfonamide drugs and penicillin and streptomycin are now available. In conformance with its policy of withdrawing acceptance of a toxic drug when a less toxic but equally effective agent becomes available, the Council voted to omit sulfathiazole and sulfathiazole sodium from the 1949 edition of New and Nonofficial Remedies."

CASE REPORTS

SPONTANEOUS MEDIASTINAL EMPHYSEMA

WYLIE F. CREEL, M.D., Austin, Texas

ONE of the physical signs of mediastinal emphysema or pneumomediastinum was described by Laennec in his book on auscultation in 1831, and a fairly adequate description of the pathogenesis of the condition was written by Müller in 1888, yet it was rarely mentioned in the literature prior to 1934. In that year Louis Hamman,[5] in the first of his papers on the subject, reported several cases, described the diagnostic precordial crunching sound, usually referred to as Hamman's sign,[6] and pointed out most of the characteristics of the condition. He was primarily interested in those so-called spontaneous cases occurring with little or no obvious cause as opposed to those secondary to childbirth, chest injuries, operations, foreign bodies, atelectasis of the newborn, and so forth. Since that time an increasing number of cases have been reported. Fagin and Schwab[3] in 1946 summarized the 36 cases in the literature and added 3 of their own. An additional 34 cases, including the 3 here summarized, have been reported since then.[2, 7, 8, 10, 11, 15, 16, 17, 18]

CLINICAL FEATURES AND COURSE

The outstanding features of this condition are chest pain, the auscultatory finding of precordial crunching, the sudden inexplicable onset in healthy young adults with predilection for the male sex, usually a benign course with recovery in a week or so, frequent occurrence with pneumothorax and subcutaneous emphysema, and a tendency toward recurrence.

The pain which can be classified as mild to moderate in most instances is located either substernally, precordially, in the left, or rarely in the right side of the chest. It frequently radiates to the neck, left shoulder, or back. Less commonly pain is referred down the left arm and rarely down the right arm. At times, especially when severe, it may easily be confused with the pain of myocardial infarction.

Hamman's sign has been described as a crackling, bubbling, crunching noise and compared to sounds produced by walking on dry snow, wrinkling cellophane, or dropping peas on taut canvas. It is characteristically loud, may even be heard at a distance of several feet, and is usually sufficiently characteristic to establish the diagnosis. Frequently it is only heard with the patient in the left lateral position. Full inspiration occasionally intensifies it; again it may be louder after expiration. It usually persists throughout both systole and diastole, with systolic intensification. The sound is presumably produced by the beating of the heart against bubbles of air in the anterior mediastinum. Examination of the chest will reveal pneumothorax in 60 per cent, almost always, although not invariably, as has been stated, on the left. Subcutaneous emphysema is noted in 15 per cent. In most cases, the presence of air renders the precordium hyperresonant. The patients usually experience increased pain when lying on the left side, regardless of the presence or absence of pneumothorax.

The onset is usually sudden and without obvious cause, although, as will be mentioned later, this may be more apparent than real. There is no good explanation for the higher incidence, 10 to 1, in males. The average age at onset is 25. It is not more common in those doing hard work day after day. The frequency of cases, 7, occurring in physicians and medical students would seem best explainable by a greater awareness of the condition.

The great majority of these patients are promptly relieved by bed rest and perhaps a dose or two of codeine. At times, if intramediastinal pressure rises sufficiently, dyspnea, cyanosis, and shock appear. Most of these cases will decompress themselves by developing subcutaneous emphysema, pneumoretroperitoneum, or pneumothorax. It is in those critically ill patients requiring surgical decompression and most resembling coronary occlusion that diagnosis is imperative. Dramatic relief by operative release of air has been reported in 2 cases.[8, 18] No deaths have been reported so far in spontaneous pneumomediastinum.

If it were not for its unpredictable course and the undesirable effects of exercise and coughing, many of these patients would hardly require hospitalization.

From the Student Health Service, The University of Texas.
Read before the Section on Medicine, State Medical Association of Texas, Annual Session, San Antonio, May 3, 1949.

In fact, many wait several days before consulting a physician. At all events, the usual patient may be discharged in a week or so, with moderate restriction of activity. While by no means invariable, the prior or subsequent occurrence of 35 pneumothoraces and/or attacks of pneumomediastinum in 17 persons does indicate the tendency to recurrence.

LABORATORY FINDINGS

The most important laboratory aid to diagnosis is the roentgen ray. It may reveal a pneumothorax or air in the mediastinum itself. This latter may only be demonstrated in oblique or lateral views, or as translucent bands bordering the heart and slightly widening the upper mediastinum in the postero-anterior film.

Various minor electrocardiographic changes—low voltage, low voltage in lead I, slight right or left axis deviation, right axis strain, low R_4, inverted T_2, and slight elevation of ST_2 and ST_3—have been reported, although most patients on whom cardiograms were made showed normal tracings. Hamman[7] reported a case in which the T wave was inverted in all limb leads with subsequent return to normal. It has been stated that these changes are due to pneumothorax, to pulmonary perivascular air infiltration, or to the muffling effect of mediastinal air per se. At all events, they are almost always minor and not likely to be confused with anything else. It seems probable, as suggested by Miller[14] and demonstrated by Littman[11] in 2 cases, that the changes are positional rather than due to any interference with coronary circulation. In most cases the routine laboratory findings are normal unless there is an infection of the upper respiratory tract.

DIFFERENTIAL DIAGNOSIS

Aside from the failure to demonstrate mediastinal air in cases treated for subcutaneous emphysema or pneumothorax, the conditions which may be confused with pneumomediastinum are myocardial infarction and acute pericarditis. In those patients having sufficient pain to suggest infarction, physical examination, roentgenograms, and cardiograms can hardly fail to establish the diagnosis. At times, especially if Hamman's sign is not loud and a coincident respiratory infection is present, it may be difficult to distinguish between pericarditis and mediastinal air. Repeated examinations in various positions, especially the left lateral, roentgenograms, and cardiograms should eventually make the distinction.

TREATMENT

Most patients need nothing more than limitation of activity. A few will require morphine and oxygen. It is only the exceptional case that will require surgical relief. There is no unanimity of opinion as to the prophylactic value of chemotherapy.[2, 17] It did not seem worth while in our patients.

PATHOGENESIS

As Hamman pointed out, air may reach the mediastinum along the fascial planes of the neck, by perforation of esophagus or trachea, from the retroperitoneal space, and from the interstitial tissues of the lung. This last is by far the commonest source. Air may pass from mediastinum to the pleural cavities, but the reverse does not occur.

Macklin[12, 13] has produced pulmonary interstitial emphysema and its sequellae in cats by forcing air into a bronchus through a catheter. By proper fixation of the pulmonary tissues, he was able to demonstrate the following course of events: There was no gross rupture of alveoli, but air bubbles accumulated in the perivascular sheaths, migrated toward the hilum, coalescing as they did so, and eventually broke free into the mediastinum. Pneumothorax was always secondary to pneumomediastinum as was subcutaneous emphysema and pneumoretroperitoneum. Overdistention of the removed trachea and lungs of recently killed calves produced pulmonary interstitial emphysema with eventual escape of the air from the vascular sheath about the hilum. Dogs placed in low pressure chambers but breathing air at atmospheric pressure developed those changes produced by overinflation in cats. A few properly fixed human autopsy cases have shown perivascular air infiltration and pneumomediastinum.

Macklin pointed out that two factors favor the flow of air from alveoli into the perivascular sheaths, regardless of intra-alveolar pressure. The first is overinflation of the alveoli without corresponding expansion of the vascular lumen. The second is a decrease in size of the contents of the perivascular sheath without diminution in the surrounding alveoli. Increased intra-alveolar pressure is practically always associated with one or both of these factors.

In those persons developing pulmonary interstitial emphysema and pneumomediastinum without apparent cause, several factors may be involved. Small areas of atelectasis with surrounding hyperinflated alveoli and congenitally defective tissues have been prominently mentioned.[7] In 6 families multiple cases have been reported.

Macklin and Macklin suggested three reasons for apparent spontaneous rupture. One, more or less hypothetical, is that of a sudden respiratory effort. Another, perhaps more attractive, is that coughing or unusual exertion some hours before may have been forgotten by the time air collects in the mediastinum in sufficient amount to cause pain or produce pneumothorax or subcutaneous emphysema. Some patients

will complain of, or later remember, a milder pain in the lateral portion of the chest some hours prior to the appearance of more severe substernal distress. This pain is likely a result of pulmonary interstitial emphysema.[13] The third possibility, especially when the condition develops during the night, is that of relative atelectasis of dependent portions of the lung and hyperinflation of other parts. The Macklins finally concluded, however, that alveolar walls which are constitutionally susceptible or weakened because of inflammation must be present in the spontaneous cases. This has not been demonstrated.

Because of the frequency of spontaneous pneumothorax, estimated at 1 a year per 1,000 at college age,[1] and its frequent association with mediastinal emphysema, their relationship is of importance. Tuberculosis, once considered of major importance in the etiology of pneumothorax, is no longer so regarded. A few cases are due to rupture of cysts or emphysematous blebs. Experimentally, pneumomediastinum causes pneumothorax and not vice versa. In Dickie's report pneumomediastinum occurred with or without pneumothorax in the same frequency. Pneumothorax without demonstrable pneumomediastinum occurred only half as frequently as did all types of pneumomediastinum. She further pointed out, as is obviously true in many cases, that Hamman's sign[9] and roentgen-ray evidence[2] are not always demonstrable, and that mediastinal air is sometimes quickly reabsorbed. Failure to examine the patient in various positions may result in failure to hear the characteristic crunch. It seems probable that more cases of pneumothorax are secondary to pulmonary interstitial emphysema and pneumomediastinum than to any other cause.

CASE REPORTS

CASE 1.—A 20 year old male student reported to the Health Service on July 17, 1947, complaining of pain in the right side of the chest for eighteen hours. Although there was a past history of asthma there was no obvious cause for the right pneumothorax which was noted clinically and in the roentgen-ray film. The patient had a temperature of 99.4 F. but was not considered in need of hospitalization. After four days all symptoms and signs disappeared.

The patient returned August 8, complaining of mild pain in the precordium of one day's duration, again without any apparent cause. In the interval he had had some brief, sharp pains in the left portion of the chest about a week before, lasting only a few hours, at which time he had not seen a doctor. Physical examination revealed a tall, slender boy, neither dyspneic nor obviously ill. The left border of cardiac dullness could not be satisfactorily percussed. The blood pressure was 120/80 and the lungs were clear. In the supine position a faint rubbing sound was heard over the heart; this changed to the crunching, grating sound of pneumomediastinum after the patient turned on his left side. The temperature was 99.4 F. All laboratory studies were normal, including chest roentgenograms, which showed complete absorption of air from the right pleural cavity. No mediastinal air could be demonstrated in oblique films nor in several follow-up postero-anterior films. Serial electrocardiograms showed low voltage in the first lead, which was not believed to be abnormal in a person of his habitus.

After three days of bed rest, during most of which time he was free of pain, the patient returned to classes. On August 11 there were a few asthmatic breath sounds but never any subjective wheezing. Daily examinations showed marked variation in the abnormal precordial noises. The most common finding was that noted on the first examination—loud crunching sounds precordially in the left lateral decubitus position and a faint rub or no adventitious sounds in other positions. At other times the only finding was a soft rubbing sound, indistinguishable from that of acute pericarditis, heard only when the patient was lying on the left side. Less commonly, Hamman's sign could be heard even when the boy was supine but never when he was sitting up. Once, on August 18, nothing abnormal could be made out in any position. The patient was asymptomatic for the most part. On August 13 and 18 there was some mild precordial distress. On August 21 he first noted a popping, gurgling sensation over the heart without pain. When leaning forward or lying on the left side at times he heard crackling sounds in his chest. On August 23, mild pain recurred and lasted five days, on the last of which examination revealed that the crunching, crackling sound was louder than at any previous time. The next day, forty-three days after the onset of the pneumothorax, pain was absent and the abnormal sounds were heard for the last time. A follow-up letter on December 7, 1948, indicated that the patient had had no further trouble.

This case is unusual in that the pneumothorax occurred on the right side, the third case so reported. Whether pneumomediastinum existed at onset is unproved but probable. The patient was not then examined either supine or when lying on the left side. The long course and marked variation from day to day in the precordial sounds with absence of symptoms most of the time is uncommon. It is possible that the wheezing heard on one examination was significant in the production of this condition. The possibility of confusion with pericarditis at times was great.

CASE 2.—A 20 year old boy was admitted to the hospital November 13, 1947, because of dyspnea and substernal pain. He had had one or two day bouts of asthma about every six months for years, the most recent of which had begun that morning. However, it had not been severe enough to prevent his participation in a rehearsal for a musical show about three hours before admission. After considerable singing he noted that his breathing was more labored and pain soon appeared and was moderately severe. Physical examination revealed dyspnea, tight asthmatic breath sounds, and wheezes throughout both lung fields. It was impossible satisfactorily to percuss the left border of cardiac dullness. There was a crepitant swelling from mastoid process to clavicle bilaterally. The temperature was 101.2 F. By morning the patient was afebrile and much more comfortable. Eighteen hours after entry Hamman's sign was first noted. It was easily heard in the supine position and was intensified by turning the patient on his left side. A roentgenogram revealed both subcutaneous and mediastinal emphysema but no pneumothorax.

His course was one of rapid improvement. Discomfort ceased the day after admission, and two days later the lungs

were clear of wheezes. By the third hospital day the precordial crunch could be demonstrated only with the patient on his left side, and the subcutaneous emphysema had largely disappeared. Assuming the left lateral decubitus caused return of slight pain. All signs had disappeared by November 19, and the patient was discharged one week after entry entirely asymptomatic. Follow-up observations indicated no further chest pain, despite one mild asthmatic attack in January, 1949.

This patient presented the syndrome of subcutaneous emphysema and mediastinal air, demonstrable clinically and by roentgen ray, precipitated by bronchial asthma plus considerable singing. The location and degree of pain was suggestive of myocardial infarction.

CASE 3.—The third patient, a man aged 30, had had a left pneumothorax in September, 1947, attributed to bending forward suddenly. Pain in the left side of the chest lasted for two weeks. Physical examination and a chest roentgenogram on March 3, 1948, were both negative. On May 11 he returned with mild pain of a few hours' duration in the left side of the chest and left lumbar regions. This was aggravated by bending and by his slight nonproductive cough. Temperature and physical examination were normal. A chest roentgenogram revealed a small pneumothorax on the left. When the patient was reexamined a few hours later, it was possible to hear loud precordial crunching, grating sounds. These were heard only with the patient lying on his left side. There was no subcutaneous emphysema nor could air be demonstrated in the mediastinum by roentgenograms taken from various angles. On direct questioning the patient stated that he felt a peculiar gurgling sensation beneath the lower sternum and in the left portion of the chest anteriorly, presumably synchronous with the heart beat. This was not particularly annoying, and he had therefore not complained of it.

The patient was much improved two days later, and on May 17 had entirely recovered. In a follow-up letter he reported that three weeks later there had been a return of mild pain in the left side of the chest lasting four days but that he had been well since then.

This case follows a frequent pattern of recurrent pneumothorax, once with, and once without, recognized pneumomediastinum. Hamman's sign was heard only in the left lateral position. The patient was aware of the disturbance caused by the beating of the heart against the mediastinal air bubbles. He did not feel nor look ill and barely had sufficient discomfort to consult a physician.

SUMMARY

The condition of spontaneous mediastinal emphysema, first adequately described by Hamman in 1934, has been reported with increasing frequency in the past fifteen years. The experimental work of Macklin and clinical observations tend to confirm the current view that the air in the mediastinum originates in alveoli subjected to increased pressure or in which the gradient favors migration of air into the perivascular tissues and thence along the blood vessels into the mediastinum. Mediastinal emphysema, pneumoretroperitoneum, subcutaneous emphysema, and pneumothorax are considered to be secondary to the initial pulmonary interstitial emphysema. Whether or not there is some congenital weakness of the alveolar or subalveolar tissues is uncertain. It appears possible that the factors mentioned above as favoring the migration of air are operable not only in those cases with an obvious cause but also in those cases of apparently spontaneous origin. The relationship of pneumomediastinum to pneumothorax, both clinically and experimentally, would indicate that the former is always primary when they coexist and that greater care in searching for air in the mediastinum would be rewarding in spontaneous pneumothorax. Hamman's sign, the typical crunching precordial sound, frequently heard only when the patient is on the left side, and the value of roentgen rays are emphasized in the differentiation of mediastinal emphysema from myocardial infarction and acute pericarditis. The condition is usually benign but may be recurrent and at times increased mediastinal pressure may require surgical relief.

CONCLUSIONS

Spontaneous mediastinal emphysema presents a characteristic clinical and roentgen-ray picture which makes the diagnosis possible in most cases. It is secondary to migration of air in the perivascular sheaths from the alveoli to the mediastinum. Overinflation, increased intra-alveolar pressure, or decrease in blood supply to the lungs are etiologic factors. There may be an inherited weakness of the alveolar or subalveolar tissues.

Although pneumomediastinum as a rule is a relatively benign condition, at times it may imitate acute pericarditis or coronary thrombosis. Rarely, surgical withdrawal of air is imperative.

From experimental and clinical experience it appears that most cases of spontaneous pneumothorax are secondary to pulmonary interstitial emphysema and mediastinal emphysema.

The 3 cases of spontaneous mediastinal emphysema here reported bring the total in the literature to 73. One of the cases here is the third to be reported in association with a right pneumothorax.

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ABSTRACT OF DISCUSSION

DR. JOHN CHAPMAN, Dallas: Dr. Creel's discussion is based upon the currently popular idea of the pathogenesis of spontaneous pneumothorax and pneumomediastinum, which was inclusively studied by Macklin. I believe a divergent point of view, which is in the minority, is justified by the following facts:

1. The crunching sound as described by Hamman is not pathognomonic of pneumomediastinum but may be encountered in patients with both spontaneous and therapeutic pneumothoraces, especially if the air is in the left pleural cavity. Furthermore, on rare occasion a similar sound may be encountered in bullous emphysema which involves the anterior dependent parts of the lungs.

2. According to the frank confession of numerous good radiologists, the roentgen-ray signs of small amounts of mediastinal emphysema are so dubious that these specialists are unwilling to report or to accept such a diagnosis and reserve it for those instances in which the mediastinum is obviously widened, in which streaks of radiolucency are evident, and in which cervical subcutaneous emphysema is in evidence.

3. Autopsy material in spontaneous pneumothorax is scarce and is mostly twenty years or more old. In all the available material, with one or two exceptions in which no possible source of air could be discovered, the findings were either bullae on the surface of the lungs or pleural blebs. Pathologic studies of 8 cases surgically explored for repeated spontaneous pneumothorax have revealed bullae or blebs in all but 1, in which no point of air escape could be found at all. Only 1 case of spontaneous pneumomediastinum, that of Oswald Jones, appears in the literature, and in that case there was no pneumothorax.

4. With more time and considerable discussion it is possible to show that Macklin's ideas, insofar as pneumothorax is concerned, are mechanically and physically contradictory. His work loses validity from the attempt to be too inclusive but, I think, is reliable in regard to the pathogenesis of real spontaneous pneumomediastinum.

From these facts it may be said that if pneumomediastinum is to be considered, pneumothorax should be shown to be absent if the diagnosis is to be based upon a crunch. For this purpose it is necessary to have films made at full expiration since small pneumothoraces may be difficult to visualize in standard inspiration films. If pneumothorax is also present, there must be acceptable radiologic signs—that is, there must be something more in evidence than mere prominence of the aortic knob or unusual delineation of the pulmonary artery or complete radiolucency along the heart margin, since all these signs likewise result from pneumothorax.

I wish again to emphasize that these views are definitely those of the minority and that the signs and symptoms which Dr. Creel has used are at present widely accepted.

NEWS

COMING MEETINGS AND CLINICS

State Medical Association of Texas, Fort Worth, May 2-4, 1950. Dr. G. V. Brindley, Temple, Pres.; Dr. Harold M. Williams, 700 Guadalupe St., Austin, Secy.

American Medical Association, Clinical Session, Washington, D. C., Dec. 6-9, 1949. Dr. Ernest E. Irons, Chicago, Pres.; Dr. George F. Lull, 535 North Dearborn St., Chicago 10, Secy.

NATIONAL AND REGIONAL

American Academy of Allergy. Dr. Will C. Sprain, New York, Pres.; Dr. Theodore L. Squier, 424 E. Wisconsin Ave., Milwaukee, Secy.

American Academy of Dermatology and Syphilology, Chicago, Dec. 3-8, 1949. Dr. Francis E. Senear, Chicago, Pres.; Dr. Earle D. Osborne, 71 North Street, Buffalo, N. Y., Secy.

American Academy of General Practice, St. Louis, Feb. 20-23, 1950. Dr. Elmer C. Texter, Detroit, Pres.; Mr. Mac F. Cahal, 406 W. 34th St., Kansas City 2, Executive Secy.

American Academy of Neurological Surgery. Dr. William S. Keith, Toronto, Canada, Pres.; Dr. T. C. Erickson, 1300 University Ave., Madison 5, Wis., Secy.

American Academy of Ophthalmology and Otolaryngology. Dr. Conrad Berens, New York, Pres.; Dr. W. L. Benedict, 100 First Ave. Bldg., Rochester, Minn, Secy.

American Academy of Pediatrics. Dr. Warren R. Sission, Boston, Pres.; Dr. C. G. Grulee, 636 Church St., Evanston, Ill., Secy.

American Association for Thoracic Surgery, Denver, Colo., April 15-19, 1950. Dr. Edward J. O'Brien, Detroit, Pres.; Dr. Brian Blades, 901 23rd St. N. W., Washington, D. C., Secy.

American Association of Genito-Urinary Surgeons, Hershey, Pa., May 26-28, 1950. Dr. J. A. C. Colston, Baltimore, Pres.; Dr. Norris J. Heckel, 122 S. Michigan Ave., Chicago 3, Secy.

American Association of Obstetricians, Gynecologists, and Abdominal Surgeons, Hot Springs, Va., Sept. 7-9, 1950. Dr. S. A. Cosgrove, Jersey City, N. J., Pres.; Dr. L. A. Calkins, University of Kansas Medical Center, Kansas City 3, Secy.

American Cancer Society. Dr. Clifford C. Nesselrode, Kansas City, Kan., Pres.; Mr. Charles D. Hilles, 47 Beaver St., New York, Secy.

American College of Physicians, Boston, April 17-21, 1950. Dr. Reginald Fitz, Boston, Pres.; Mr. E. R. Loveland, 4200 Pine St., Philadelphia 4, Secy.

American College of Radiology, San Francisco, June 25, 1950. Dr. Arthur W. Erskine, Cedar Rapids, Iowa, Pres.; Mr. W. C. Stronach, 20 N. Wacker Drive, Chicago 6, Secy.

American College of Surgeons. Dr. Frederick A. Coller, Ann Arbor, Mich., Pres.; Dr. Paul B. Magnuson, 40 E. Erie St., Chicago 11, Secy.

American Congress of Physical Medicine, Boston, Aug. 29-Sept. 1, 1950. Dr. Earl C. Elkins, Rochester, Minn., Pres.; Dr. Richard Kovacs, 2 E. 88th St., New York 28, Secy.

American Dermatological Association, Jasper National Park, Canada, June 18-22, 1950. Dr. Charles C. Dennie, Kansas City, Mo., Pres.; Dr. L. A. Brunsting, 102 2nd Ave., S. W., Rochester, Minn., Secy.

American Gastro-Enterological Association, Atlantic City, April 28-29, 1950. Dr. J. Arnold Bargen, Rochester, Minn., Pres.; Dr. Dwight L. Wilbur, 655 Sutter St., San Francisco, Secy.

American Gynecological Society, White Sulphur Springs, W. Va., May 11-13, 1950. Dr. Ludwig Emge, San Francisco, Pres.; Dr. Howard Taylor, 842 Park Ave., New York 21, Secy.

American Hospital Association. Mr. Joseph G. Norby, Milwaukee, Pres.; Mr. George P. Bugbee, 18 E. Division St., Chicago, Executive Secy.

American Laryngological, Rhinological, and Otological Society, San Francisco, May 25-27, 1950. Dr. Robert C. Martin, San Francisco, Pres.; Dr. C. S. Nash, 277 Alexander St., Rochester 7, N. Y., Secy.

American Neurological Association, Atlantic City, June 12-14, 1950. Dr. Henry W. Woltman, Rochester, Minn., Pres.; Dr. H. Houston Merritt, 710 W. 168th St., New York 32, Secy.

American Ophthalmological Society, Hot Springs, Va., May 31-June 2, 1950. Dr. Parker Heath, Boston, Pres.; Dr. M. C. Wheeler, 30 W. 59th St., New York 19, Secy.

American Orthopedic Association, Virginia Beach, Va., May, 1950. Dr. R. W. Johnson, Jr., Baltimore, Pres.; Dr. C. Leslie Mitchell, Henry Ford Hospital, Detroit 2, Secy.

American Pediatric Society, French Lick, Ind.. May 8-10, 1950. Dr. Phillip C. Jeans, Iowa City, Pres.; Dr. Henry G. Poncher, 1819 W. Polk St., Chicago 12, Secy.

American Proctologic Society. Dr. Louis E. Moon, Omaha, Pres.; Dr. W. Wendell Green, 1838 Parkwood Ave., Toledo 2, Secy.

American Psychiatric Association, Detroit, May 1-5, 1950. Dr. George S. Stevenson, New York, Pres.; Dr. Leo H. Bartemeier, General Motors Bldg., Detroit, Secy.

American Public Health Association. Dr. Charles F. Wilinsky, Boston, Pres.; Dr. R. M. Atwater, 1790 Broadway, New York 19, Secy.

American Society of Anesthesiologists, New York, Dec. 7-10, 1949. Dr. H. Boyd Stewart, Tulsa, Okla., Pres.; Dr. Curtis B. Hickcox, 188 W. Randolph St., Chicago, Secy.

American Society of Clinical Pathologists. Dr. Osborne A. Brines, Detroit, Pres.; Dr. Clyde G. Culbertson, Indiana University School of Medicine, Indianapolis, Secy.

American Surgical Association, Colorado Springs, April 19-23, 1950. Dr. Thomas Orr, Kansas City, Kan., Pres.; Dr. Nathan Womack, University of Iowa, Iowa City, Secy.

American Urological Association, Washington, D. C., May 29-June 1, 1950. Dr. Carl F. Rusche, Hollywood, Calif., Pres.; Dr. C. H. deT. Shivers, 121 S. Illinois Ave., Atlantic City, N. J., Secy.

International College of Surgeons, U. S. Chapter. Dr. Curtis L. Hall, Washington, D. C., Pres.; Dr. Arnold S. Jackson, 1516 Lake Shore Dr., Chicago, Secy.

National Tuberculosis Association, Washington, D. C., April 25-28, 1950. Dr. R. D. Thompson, La Vina, Calif., Pres.; Dr. H. Stuart Willis, 1790 Broadway, New York 19, Secy.

Radiological Society of North America, Cleveland, Dec. 4-9, 1949. Dr. Edgar P. McNamee, Cleveland, Pres.; Dr. D. S. Childs, Medical Arts Bldg., Syracuse 2, N. Y., Secy.

Southern Medical Association. Dr. Hamilton W. McKay, Charlotte, N. C., Pres.; Mr. C. P. Loranz, 1020 Empire Bldg., Birmingham, Ala., Secy.

Southern Psychiatric Association. Dr. Walter J. Otis, New Orleans, Pres.; Dr. Newdigate M. Owensby, Medical Arts Bldg., Atlanta, Ga., Secy.

Southern Surgical Association, Hot Springs, Va., Dec. 6-8, 1949. Dr. Alfred Blalock, Baltimore, Pres.; Dr. John C. Burch, 2112 West End Ave., Nashville, Tenn., Secy.

Southwest Allergy Forum, Memphis, Tenn., April, 1950. Dr. L. O. Dutton, El Paso, Pres.; Dr. Sam Sanders, 1089 Madison Ave., Memphis, Tenn., Secy.

Southwest Regional Cancer Conference. Secy., 209 Medical Arts Bldg., Fort Worth.

Southwestern Medical Association, Phoenix, Ariz., 1950. Dr. I. J. Marshall, Roswell, N. Mex., Pres.; Dr. W. W. Schuessler, 1415 First National Bank Bldg., El Paso, Secy.

Southwestern Surgical Congress, Kansas City, Sept., 1950. Dr. Thomas G. Orr, Kansas City, Pres.; Dr. C. R. Rountree, 210 Plaza Court, Oklahoma City 3, Secy.

Tri-State Medical Assembly, Shreveport, La., 1950. Dr. Charles Gowen, Shreveport, Pres.; Dr. John Walter Jones, 401 E. Fifth St., Texarkana, Secy.

United States-Mexico Border Public Health Association, Chihuahua, 1950. Dr. George W. Cox, Austin, Pres.; Dr. M. F. Haralson, 314 U. S. Court House, El Paso, Secy.

STATE

Texas Academy of General Practice, Fort Worth, Sept. 25, 1950. Dr. H. T. Jackson, Fort Worth, Pres.; Dr. W. P. Higgins, Jr., Medical Arts Bldg., Fort Worth, Secy.

Texas Air-Medics Association, Fort Worth, May 1, 1950. Dr. C. Hansford Brownlee, Austin, Pres.; Dr. C. F. Miller, 906 Medical Arts Bldg., Waco, Secy.

Texas Association of Obstetricians and Gynecologists, San Antonio, Feb. 3-4, 1950. Dr. Howard Smith, Marlin, Pres.; Dr. George F. Adam, 4115 Fannin, Houston, Secy.

Texas Chapter, American College of Chest Physicians, Fort Worth, May 1, 1950. Jesse B. White, Amarillo, Pres.; Dr. Henry R. Hoskins, 514 Medical Arts Bldg., San Antonio, Secy.

Texas Club of Internists. Dr. W. Shelton Barcus, Fort Worth, Pres.; Dr. Victor E. Schulze, 219 S. Magdalen St., San Angelo, Secy.

Texas Dermatological Society, Fort Worth, May 1, 1950. Dr. A. G. Schoch, Dallas, Pres.; Dr. W. Harris Connor, 601 Medical Arts Bldg., Houston, Secy.

Texas Diabetes Association, Fort Worth, April 30, 1950. Dr. J. Shirley Sweeney, Gainesville, Pres.; Dr. W. N. Powell, W. Ave. F, Temple, Secy.

Texas Division, American Cancer Society. Mr. Frank C. Smith, Houston, Pres.; Mrs. Jack Hutchins, El Campo, Secy.

Texas Heart Association, Fort Worth, May 1, 1950. Dr. Merritt B. Whitten, Dallas, Pres.; Miss Roberta Miller, Medical Arts Bldg., Dallas, Executive Secy.

Texas Hospital Association, Galveston, March 7-9, 1950. Mr. Julian H. Pace, Waco, Pres.; Mrs. Ruth Barnhart, 2210 Main St., Dallas, Secy.

Texas Neuropsychiatric Association, Fort Worth, May 1, 1950. Dr. A. T. Hanretta, Austin, Pres.; Dr. David Wade, 510 Capital National Bank Bldg., Austin, Secy.

Texas Orthopedic Association, Fort Worth, May 1, 1950. Dr. Bruce Stephenson, Beaumont, Pres.; Dr. Margaret Watkins, 3629 Fairmount St., Dallas, Secy.

Texas Pediatric Society, Fort Worth, Oct. 6-7, 1950. Dr. J. E. Ashby, Dallas, Pres.; Dr. M. C. Carlisle, 1410 Austin Ave., Waco, Secy.

Texas Public Health Association, Galveston, Feb. 21-24, 1950. Dr. W. R. Ross, Tyler, Pres.; Mr. Earle W. Sudderth, Dallas County Health Department, Court House, Dallas, Executive Secy.

Texas Radiological Society, Dallas, Feb. 3-4, 1950. Dr. J. J. Faust, Tyler, Pres.; Dr. R. P. O'Bannon, 650 Fifth Ave., Fort Worth, Secy.

Texas Railway and Traumatic Surgical Association, Fort Worth, May 1, 1950. Dr. Joe Gandy, Houston, Pres.; Dr. W. F. Parsons, First National Bank Bldg., Fort Worth, Secy.

Texas Rheumatism Associataion, Fort Worth, May 1, 1950. Dr. Howard C. Coggeshall, Dallas, Pres.; Dr. Robert H. Mitchell, 210 Medical Arts Bldg., Fort Worth, Secy.

Texas Society for Mental Hygiene, Mineral Wells, April 13-14, 1950. Dr. Arthur Schwenkenberg, Dallas, Pres.; Mrs. Elizabeth F. Gardner, 1617 Watchhill Road, Austin 21, Executive Secy.

Texas Society of Anesthesiologists, Fort Worth, May 1, 1950. Dr. Wilbur F. Robertson, San Antonio, Pres.; Dr. H. C. Slocum, 928 Strand, Galveston, Secy.

Texas Society of Gastroenterologists and Proctologists, Fort Worth, May 1, 1950. Dr. G. E. Brereton, Dallas, Pres.; Dr. John S. Bagwell, Medical Arts Bldg., Dallas, Secy.

Texas Society of Ophthalmology and Otolaryngology, San Antonio, Dec. 2-3, 1949. Dr. August J. Streit, Amarillo, Pres.; Dr. John L. Matthews, 929 Nix Professional Bldg., San Antonio, Secy.

Texas Society of Pathologists. Dr. John F. Pilcher, Corpus Christi, Pres.; Dr. A. O. Severance, 205 Camden, San Antonio, Secy.

Texas Surgical Society, Dallas, April 3-4, 1950. Dr. R. J. White, Fort Worth, Pres.; Dr. Truman G. Blocker, 927 Strand, Galveston, Secy.

Texas Tuberculosis Association, Austin, April 7-8, 1950. Dr. Elliott Mendenhall, Dallas, Pres.; Miss Pansy Nichols, 208 E. Ninth, Austin, Executive Secy.

Texas Urological Society, San Antonio. Dr. Hub E. Isaacks, Fort Worth, Pres.; Dr. John M. Pace, 428 Medical Arts Bldg., Dallas, Secy.

DISTRICT

Second District Society. Dr. Charles E. Britt, Midland, Pres.; Dr. Robert M. Golladay, 1203 W. Wall St., Midland, Secy.

Third District Society, Amarillo, April, 1950. Dr. C. B. Jones, Wellington, Pres.; Dr. Roy G. Loveless, 2609 Nineteenth St., Lubbock, Secy.

Fourth District Society, Brownwood. Dr. Gordon F. Madding, San Angelo, Pres.; Dr. S. B. Locker, First National Bank Bldg., Brownwood, Secy.

Fifth and Sixth Districts Society, Corpus Christi, July 7-8, 1950. Dr. Franklin W. Yeager, Corpus Christi, Pres.; Dr. Foy Moody, 1611 Fifth St., Corpus Christi, Secy.
Seventh District Society. Dr. David Wade, Austin, Pres.; Dr. Joe W. Bailey, 1411 San Antonio St., Austin, Secy.
Eighth District Medical Society, Galveston, March 10-11, 1950. Dr. Leonard Johnson, El Campo, Pres.; Dr. Robert Casey, Texas City, Secy.
Tenth District Medical Society. Dr. J. A. Richardson, Sr., Jasper, Pres.; Dr. Dale H. Davies, Liberty, Secy.
Eleventh District Society. Dr. E. G. Faber, Tyler, Pres.; Dr. John Travis, Jacksonville, Secy.
Twelfth District Society, Waco, Jan. 10, 1950. Dr. J. C. Terrell, Stephenville, Pres.; Dr. H. F. Connally, Jr., Amicable Bldg., Waco, Secy.
Thirteenth District Society, Mineral Wells, Oct., 1950. Dr. R. L. Daily, Wichita Falls, Pres.; Dr. S. W. Wilson, Medical Arts Bldg., Fort Worth, Secy.
Fourteenth District Society. Dr. J. Shirley Sweeney, Gainesville, Pres.; Dr. L. W. Johnson, 502 W. College St., Terrell, Secy.
Fifteenth District Society, Texarkana, 1950. Dr. F. V. Mondrik, Marshall, Pres.; Dr. Hardy Cook, Longview, Secy.

CLINICS

Dallas Southern Clinical Society, Dallas, March 13-16, 1950. Miss Betty Elmer, Medical Arts Bldg., Dallas 1, Executive Secy.
International Post-Graduate Medical Assembly of Southwest Texas, San Antonio, Jan. 24-26, 1950. Dr. John J. Hinchey, 643 Moore Bldg., San Antonio 5, Secy.
New Orleans Graduate Medical Assembly, New Orleans, March 6-9, 1950. Dr. Woodard D. Beacham, Room 105, 1430 Tulane Ave., New Orleans 12, Secy.
North Texas-Southern Oklahoma Fall Clinical Conference, Wichita Falls. Dr. James T. Lee, Wichita Falls Clinic Hospital, Wichita Falls, Program Chairman.
Oklahoma City Clinical Society Conference, Oklahoma City. Mrs. Muriel R. Waller, 512 Medical Arts Bldg., Oklahoma City 2, Executive Secy.
Post Graduate Medical Assembly of South Texas, Houston, Nov. 29-Dec. 1, 1949. Dr. E. Trowbridge Wolf, 229 Medical Arts Bldg., Houston, Secy.

UNIVERSITY OF TEXAS MEDICAL BRANCH

Two faculty members of the University of Texas Medical Branch, Galveston, Dr. Charles R. Allen, associate professor of anesthesiology, and Dr. William C. Levin, associate professor of internal medicine, have been appointed as consultants to the medical division of the Oak Ridge Institute of Nuclear Studies, Oak Ridge, Tenn., reports the Galveston *News*.

A Sigma Xi chapter was installed October 28 at the Medical Branch. The new organization evolved from the Research Club.

A postgraduate course on tumors was offered in cooperation with the State Department of Health at the Medical Branch, November 2-5. Guest speakers included Drs. B. M. Black, chief of the Section of Surgery, Mayo Clinic, Rochester, Minn.; J. T. Farrell, Jr., professor of clinical medicine, Graduate School of Medicine, University of Pennsylvania, Philadelphia; C. A. Good, Jr., assistant professor of radiology, Mayo Foundation Graduate School of Medicine, University of Minnesota, Minneapolis; E. B. Helwig, Armed Forces Institute of Pathology; R. L. Sanders, professor of surgery, University of Tennessee College of Medicine, Memphis; and R. A. Willis, director of Pathology Department, Royal Cancer Hospital, London, England.

Recent grants to the Medical Branch include $500 by the Campbell Products, Inc., to support cardiac research under the direction of Dr. Arthur Ruskin of the Department of Internal Medicine; $1,700 by the Texas Division of the American Cancer Society to support tumor conferences under the direction of Dr. Paul Brindley, professor of pathology; and $3,000 from the Upjohn Company, $3,000 from Ciba Pharmaceutical Products, and $2,500 from Sharp and Dohme to support studies under the direction of Dr. Edgar J. Poth, professor of surgery and director of the Surgical Research Laboratory.

Dr. G. W. N. Eggers, professor of orthopedic surgery at the Medical Branch, received the Robert Danis award for surgical treatment of fractures given through the International Society of Surgery, which met in New Orleans in October, reports the University of Texas *Alumni Bulletin*.

Texas Fellows of College of Surgeons

Twenty-seven Texas physicians were received into fellowship during the closing session of the annual clinical congress of the American College of Surgeons, held in Chicago from October 21 to 23. They were among the 921 initiates and 8 honorary fellows to be received into the society.

The Texas fellows are Drs. William Ainsworth, Galveston; Ernest E. Anthony, Fort Worth; Dale J. Austin, Dallas; C. Keith Barnes, Fort Worth; Emerson K. Blewett, Austin; Romie M. Dufner, San Antonio; George Ehni, Houston; John J. Hinchey, San Antonio; Rush Q. Hunter, Palestine; Louis W. O. Janssen, Corpus Christi; Denton Kerr, Houston; Maynard D. Knight, San Angelo; Harl D. Mansur, Jr., Wichita Falls; Marion H. Morris, San Antonio; Carl A. Moyer, Dallas; Edwin L. Mueller, San Antonio; Ralph A. Munslow, San Antonio; Thomas M. Oliver, Waco; A. M. Parsons, Houston; Robert H. Rowland, Jr., Sherman; David M. Rumph, Fort Worth; Chester C. Shotts, San Antonio; Albert O. Singleton, Jr., Galveston; Douglas R. Swetland, Marlin; Edgar H. Vaughn, Tyler; H. Vincent Walker, San Antonio; and Edward Zidd, Austin.

WORLD MEDICAL ASSOCIATION

Dr. Elmer Henderson, Louisville, Ky., president-elect of the American Medical Association, was named president-elect of the World Medical Association at the Third General Assembly in London, October 10-15. Forty-two countries are represented in the World Medical Association. Dr. Henderson will succeed Dr. Charles Hill of the British Medical Association, taking office when the assembly meets in New York next October.

Dr. R. L. Sensenich, South Bend, Ind., was elected to succeed Dr. Henderson as a member of the Council of the World Medical Association.

The General Assembly adopted an International Code of Ethics with the provision that it be left to national medical associations to modify the wording for use within their respective countries, provided that it conforms to the principles of the code.

Houston Plans Hall of Health

An extensive health exhibit to be called "Hall of Health" has been planned by the Houston Museum of Natural History, reports the Houston *Chronicle*. Committee members for the exhibit are Dr. Harold A. Wood, chairman; Dr. Dan Wunderman, Dr. James Scholfield, Roger Moehlman, Lawrence Bender, Mrs. Beulah Wild, and Mrs. Edna Miner.

Topics which will be included are the cause and prevention of common diseases; vitamin deficiency; principles of immunization; current problems of public health in relation to water supply, drainage, and mosquito and rat control; food inspection; fundamentals of anatomy and physiology; and the derivation and history of medicines.

American College of Allergists to Meet

The American College of Allergists will meet January 15-18 in St. Louis with Dr. Vera Reader Walker, Oxford, England, as guest speaker. Forty-two well known physicians will speak on a variety of subjects including those relating to cosmetics, chronic urticaria, hay fever, character problems in allergic children, and marital adjustments in parents of allergic children.

PERSONALS

Dr. Phil Bleakney, Harlingen, was awarded a plaque by the South Central Section of the American Urological Association at its recent meeting in Colorado Springs, reports the *Valley Morning Star.* The award, the first to be presented for an outstanding case report, was given to Dr. Bleakney for his paper on histoplasmosis.

Dr. Ozro T. Woods, Dallas, has been named head of the Citizens Committee on Mental Health, a statewide group appointed by Governor Allen Shivers, reports the Fort Worth *Star-Telegram.*

Dr. and Mrs. P. L. Vardy, Estelline, were honored by residents of the community by a dinner and open house on their golden wedding anniversary October 9, according to the Amarillo *Globe.*

Dr. Gough Alexander, Terrell, rode his 8 year old stallion, a palomino, to first place in the Agricultural Fair horse show in Grand Saline on September 24. Sixty-five horses were entered, informs the Grand Saline *Sun.*

Dr. I. P. Sessions, Rockdale, was honored on his eighty-second birthday October 25 with a family dinner given in his home, according to the Rockdale *Reporter.*

Dr. C. M. Cash, San Benito, was awarded the thirty-third degree in Masonry, highest honorary recognition in the order, in Washington, D. C., on October 21, states an item in the Abilene *Reporter News.*

Dr. Lee Williamson, Abilene, married Miss Mary Wyley in Abilene recently.

Dr. and Mrs. D. K. Boyd, Denton, are the recent parents of a girl.

Dr. and Mrs. Carter Anderson, Jr., Tyler, recently became the parents of a boy.

BASIC SCIENCE EXAMINATIONS

An extra examination, especially for practitioners eligible for Texas licenses by reciprocity as soon as they obtain basic science certificates, has been scheduled by the Board of Examiners in the Basic Sciences for January 20 and 21 in Austin. The regular examinations will be held April 21 and 22.

Anyone interested in the basic science examinations may write the Board at 306 Nalle Building, Austin. The examination may be taken by those who complete application a week before the examination date, informs Henry B. Hardt, Ph. D., Fort Worth, president of the Board.

NATIONAL CONFERENCE ON HEART DISEASE

A National Conference on Cardiovascular Diseases will be held in Washington, D. C., from January 18 to 20, 1950, under the sponsorship of the American Heart Association and the National Heart Institute of the U. S. Public Health Service. It will be the first meeting to bring together physicians, scientists, community service leaders, and members of allied professions to plan a comprehensive program to combat heart disease.

The meeting will include representatives from the fields of nursing, social work, hospital administration, rehabilitation, public health, and others concerned with various aspects of the heart disease control program. Both immediate and long-range programs will be defined and developed by the conference. The group will also map out plans to determine how professional and lay groups concerned with heart disease can cooperate most effectively.

TEXAS PHYSICIANS MAY RECEIVE CANCER MONOGRAPHS

A series of color illustrated monographs on the early diagnosis of cancer are now being distributed free to Texas physicians. The monographs, prepared by some of the nation's leading surgeons and scientists, are published by the American Cancer Society.

The first two of the series have already been distributed and four more are now being prepared for mailing during the next several months. The monographs now being prepared are: (1) "Diagnosis of Genito-urinary Tumors," by Dr. Victor F. Marshall; (2) "Value of Early Diagnosis in Malignant Lymphomas and Leukemia," by Dr. Lloyd F. Craver; (3) "Early Diagnosis of Breast Cancer," by Dr. Cushman D. Haagensen; and (4) "Cancer of The Lung," by Dr. Richard H. Overhold.

Physicians wishing to receive the monographs may send a post card with name and address to the American Cancer Society, Texas Division, 2307 Helena Street, Houston 6.

COURSES IN CYTOLOGIC DIAGNOSIS OF CANCER

Courses in exfoliative cytology for the diagnosis of cancer by the smear technique will be offered in the near future for pathologists and qualified physicians at the University of Colorado School of Medicine, Denver. Two types of courses will be offered, one meeting one afternoon each week for twelve weeks, the other daily for two weeks. The Laboratory of Exfoliative Cytology is open for study to individual physicians by appointment.

A two weeks' course in staining technique and a four months' course in the techniques of staining and screening are now being offered for technicians by appointment.

Further information may be obtained by writing Dr. Walter T. Wikle, Director of Laboratory of Exfoliative Cytology, University of Colorado School of Medicine, 4200 East Ninth Avenue, Denver 7.

LIBRARY SECTION

PACKAGE SERVICE

The package library consists of collections of reprints and other periodical material on various subjects, prepared for lending to members of the Association. Request for packages should be addressed "Library, State Medical Association of Texas, 700 Guadalupe Street, Austin, Texas." Twenty-five cents in stamps should be enclosed with the request to cover postage and part of the expense of collecting the material. Packages are allowed to remain in the hands of the borrower for 14 days.

ACCESSIONS

The following additions were made to the Library during November:

Reprints received, 2,380.

Journals received, 281.

Books received, 25.

The Year Book of Pediatrics, by Porcher and others (editors), and *The Year Book of Radiology,* by Hodges and others (editors), from The Year Book Publishers, Inc., Chicago.

Brain Tumors and Care of the Neurological Patient (2nd edition), by Sachs; *Atlas of Amputations,* by Slocum; and *The Doctor, the Patient and the Law* (2nd edition), by Regan, from C. V. Mosby Company, St. Louis.

Studies in Pediatrics and Medical History, by Kagan and others (editors), from Froben Press, Inc., New York.

Modern Practice in Ophthalmology, by Stallard (editor), and *Vision, Its Development in Infant and Child,* by Gesell and others, from Paul B. Hoeber, Inc., New York.

Clinical Allergy (2nd edition), by Tuft, from Lea & Febiger, Philadelphia.

Acute Appendicitis and Its Complications, by Boyce, from Oxford University Press, New York.

Cystoscopy and Urography (3rd edition), by Macalpine, from Williams & Wilkins Company, Baltimore.

For Doctors Only, by Golden, from Frederick Fell, Inc., New York.

The Eye and Its Diseases (2nd edition), by Berens; *Diseases of the Heart,* by Friedberg; *Normal Values in Clinical Medicine,* by Sunderman; and *An Atlas of the Blood and Bone Marrow,* by Custer, from W. B. Saunders Company, Philadelphia.

Clinical Diagnosis by Laboratory Examination (2nd edition), by Kolmer, from Appleton-Century-Crofts, Inc., New York.

Allergy in Relation to Otolaryngology, by Hansel, and *X-Ray Treatment: Its Origin, Birth, and Early History,* by Grubbe, from Bruce Publishing Company, St. Paul and Minneapolis.

The Road Ahead, by Flynn, from The Devin-Adair Company, New York.

Modern Drug Encyclopedia (4th edition), by Howard, from Drug Publications, Inc., New York.

Transactions of the Southern Surgical Association, vol. 60, by Burch (editor), from J. B. Lippincott Company, Philadelphia.

The Case Against Socialized Medicine, by Sullivan, from The Statesman Press, Washington, D. C.

Diagnosis and Treatment of Adrenal Insufficiency, by Thorn, from Charles C. Thomas, Springfield, Ill.

Monographs on Surgery, 1950, by Carter (editor), from Thomas Nelson & Sons, New York.

SUMMARY OF SERVICE

Local users, 45.	Borrowers by mail, 125.
Items consulted, 360.	Packages mailed, 139.
Items borrowed, 121.	Items mailed, 711.

Films loaned, 51.

Total number of items consulted, borrowed, and mailed, 1,243.

LIBRARY NEEDS

The journals listed are needed by the Library of the State Medical Association to complete volumes for binding. Any of these numbers will be acceptable either as a gift or for purchase. It is preferable that the Library, 700 Guadalupe, Austin, be notified regarding items available, and the prices of such items, if any, before shipment is made.

Journals needed by the Library of the State Medical Association are as follows:

British Journal of Surgery, Vol. 27, No. 1 (Jan.), No. 2 (April) 1939.

Bulletin of John Sealy Hospital, Vol. 1, No. 1 (Feb.), No. 6 (Oct.) 1939.

Calcutta Medical Journal, Vol. 36, No. 3-6 (March-June) 1939.

Industrial Medicine, Vol. 8, No. 2 (Feb.) 1939.

Journal of International College of Surgeons, Vol. 2, No. 1-4 (Feb., April, June, Aug.) 1939.

Journal of Missouri State Medical Association, Vol. 36, No. 5 (May) 1939.

Medical Annals of District of Columbia, Vol. 8, No. 12 (Dec.) 1939.

Medical Record, Vol. 149, No. 4 (April) 1939.

Mississippi Doctor, Vol. 16, No. 1-8 (June-Jan.) 1938-1939.

Ohio State Medical Journal, Vol. 35, No. 1 (Jan.) 1939.

Pennsylvania Medical Journal, Vol. 42, No. 8 (Aug.) 1939.

Southern Medicine and Surgery, Vol. 101, No. 1-5 (Jan.-May) 1939.

Southwestern Medicine, Vol. 24, No. 1 (Jan.) 1940.

Surgery, Vol. 5, No. 4 (April) 1939.

MOTION PICTURE FILM LIBRARY

Motion picture films on medical subjects, 16 mm., both silent and sound, some in color, and suitable for either medical or lay audiences, are available for loan to county medical societies, hospital staffs, or individual physicians, on request. Borrowers will be required to pay only the cost of shipment of the films, by express, with insurance, and for any damage to films in the hands of the borrower.

Request for films should be addressed to "Motion Picture Film Library, State Medical Association of Texas, 700 Guadalupe Street, Austin, Texas." A list of available films, with descriptions, will be furnished on request.

The following motion picture films were loaned by the Film Library during November:

Accent on Use (National Foundation for Infantile Paralysis)—Alpha Epsilon Delta Pre-Medical Club, University of Texas, Austin.

Adolescence, Introduction to (Mead Johnson)—Newton County Memorial Hospital, Newton.

Anemia, Erythroblastic (Mead Johnson)—Medical Staff of the Winkler County Memorial Hospital, Kermit.

Anemias (Lederle Laboratories, Inc.)—Brackenridge Hospital School of Nursing, Austin, and Newton County Memorial Hospital, Newton.

Anesthesia, Novocain, in Obstetrics (Winthrop Chemical Company)—Staff of Newton County Memorial Hospital, Newton, and Gonzales County Medical Society, Gonzales.

Anesthesia, Regional (Winthrop Chemical Company)—Dr. T. S. Whitecloud, Newton, and Premedical Club, Baylor University, Waco.

Antitoxins, Globulin Modified (Lederle Laboratories, Inc.)—University of Houston School of Nursing, Houston, and Harris College of Nursing, Fort Worth.

Appendicitis in Childhood (Mead Johnson)—Arlington State College, Arlington.

Bleeding Tendency, Methods for Determination of (Mead Johnson)—Brackenridge Hospital School of Nursing, Austin.

Blood Transfusion (British Information Services)—Brackenridge Hospital School of Nursing, Austin.

Blood Transfusion, Technique of (Mead Johnson)—School of Nursing, Brackenridge Hospital, Austin.

Bone Marrow (Armour Laboratories)—Methodist Hospital School of Nursing, Houston, and Staff of Scott and White Clinic, Temple.

Cancer: Problem of Early Diagnosis (American Cancer Society)—Southwestern University Pre-Medical Association, Georgetown, and Alpha Epsilon Delta Pre-Medical Club, University of Texas, Austin.

Congenital Cardio-Vascular Anomalies Amenable to Surgery (Mead Johnson)—Staff of Baylor Hospital, Dallas, and Crosbyton Clinic Hospital, Crosbyton.

Cholecystectomy (Mead Johnson)—Brackenridge Hospital School of Nursing, Austin.

D. D. T., Story of (British Information Services)—Newton County Memorial Hospital Staff, Newton.

Diphtheria and Croup (Lederle Laboratories, Inc.)—University of Houston School of Nursing, Houston.

Dysmenorrhea, Primary (Searle and Company)—Newton County Hospital Staff, Newton, and University of Houston School of Nursing, Houston.

Encephalomyelitis (Lederle Laboratories, Inc.)—Harris College of Nursing, Fort Worth.

Esophagogastrostomy, Supra-Aortic, for Carcinoma of the Midportion of the Esophagus (Dr. Philip Thorek, Chicago)—Staff of Baylor Hospital, Dallas.

Eyes for Tomorrow (Hurst Eye, Ear, Nose, and Throat Clinic)—Comal Sanitarium, New Braunfels.

Folvite (Lederle Laboratories, Inc.) — Medical Staff, Winkler County Memorial Hospital, Kermit.

Forty Billion Enemies (Westinghouse Electric and Manufacturing Company)—San Antonio Academy, San Antonio.

From Moo to You (Borden Company)—Bell County Public Schools, Temple.

Gastrectomy, Safer (Billy Burke Productions)—Comal Sanitarium Staff, New Braunfels.

Golden Glory (Standard Brands, Inc.)—Bell County Public Schools, Temple, and San Antonio Academy, San Antonio.

Goodbye Mr. Germ (Texas Tuberculosis Association)—Bell County Public Schools, Temple.

Hematology, Animated (Armour Laboratories)—Dr. N. L. Schiller, Austin; Bastrop Clinic, Bastrop; Methodist Hospital School of Nursing, Houston; and Staff of Scott and White Clinic, Temple.

Hepatitis, Observation on (Mead Johnson)—Brackenridge Hospital School of Nursing, Austin.

Human Fertility (Ortho-Products, Inc.) — Cass-Marion Counties Medical Society, Jefferson.

Human Sterility (Winthrop Chemical Company)—Cass-Marion Counties Medical Society, Jefferson, and Students of University of Texas Medical Branch, Galveston.

Hypodermic Syringes and Needles: Their Care and Function (Becton, Dickinson & Company)—Newton County Memorial Hospital Staff, Newton, and Seminole General Hospital Staff, Seminole.

Immunization Against Infectious Diseases (Lederle Laboratories)—University of Houston School of Nursing, Houston.

New Horizons (National Foundation for Infantile Paralysis)—Dr. Floyd E. Guy, Big Lake.

Plain Facts (American Social Hygiene Association)—Comal Sanitarium Staff, New Braunfels.

Pneumonia (Mead Johnson)—University of Houston School of Nursing, Houston.

Polio: Diagnosis and Management (British Information Services)—Dr. Floyd E. Guy, Big Lake.

Poliomyelitis, Sister Kenny Method of Treatment of (Dr. Herbert Hipps)—Memorial Hospital School of Nursing, Houston.

Portacaval Shunt for Portal Hypertension (Dr. Philip Thorek, Chicago)—Bastrop Clinic, Bastrop.

Pregnancy, Multiple (Mead Johnson)—LaSalle-Frio-Dimmitt Counties Medical Society, Pearsall.

Premature Infant (Mead Johnson)—LaSalle-Frio-Dimmitt Counties Medical Society, Pearsall.

Preventive Medical Program for Children (Mead Johnson)—Students of University of Texas Medical Branch, Galveston.

Problem Child (Pet Milk Company)—Alamo P. T. A., Brenham.

Scarlet Fever (Lederle Laboratories, Inc.) — Memorial Hospital School of Nursing, Houston.

Techniques of Injection (Becton, Dickinson & Company)—Brackenridge Hospital School of Nursing, Austin, and Newton County Memorial Hospital Staff, Newton.

Uterosalpingography, Serial (E. Fougera & Company)—Comal Sanitarium Staff, New Braunfels.

Varicose Veins and Their Complications (Becton, Dickinson & Company)—Crosbyton Clinic Hospital, Crosbyton.

Varicose Veins, Treatment of (G. D. Searle & Company)—Students of University of Texas Medical Branch, Galveston.

When Bobby Goes to School (Mead Johnson)—Central P. T. A., Brenham.

X-Ray, Chest, Routine Admission in General Hospitals (Texas Tuberculosis Association)—South Texas Society of X-Ray Technicians, Corpus Christi.

X-Ray, Chest, Technique of Group Services (Texas Tuberculosis Association)—South Texas Society of X-Ray Technicians, Corpus Christi.

BOOK NOTICES

[1]Synopsis of Psychosomatic Diagnosis and Treatment

Flanders Dunbar, M. D. Cloth, 501 pages. Price, $6.50. St. Louis, C. V. Mosby Company, 1948.

In view of the importance of psychosomatic medicine in medical practice, this book is a timely presentation. Flanders Dunbar and her associates have given in their respective chapters a review that follows Osler's outline so as to "add a section on psychosomatic diagnosis and treatment to each chapter of Christian's revision of Osler's textbook." In addition, there is a discussion of the chronology of intrauterine development, of the determinants of predisposition to psychosomatic dysfunction, of the neuroses and their accompaniment in physical dysfunction, and of industrial medicine, pediatrics, and geriatrics in their relationship to psychosomatic medicine. This book thus covers the whole field of psychosomatic research and aims to guide the busy physician to a better understanding of the psychosomatic problems of his patients.

The author's close association with the study of psychosomatic conditions, their causation and mechanism, and the extensive coverage of the literature make this monograph a welcome addition. It is highly recommended to everyone interested in this particular aspect of medical practice.

[2]Blakiston's New Gould Medical Dictionary

Harold Wellington Jones, M. D.; Normand L. Hoerr, M. D.; and Arthur Osol, Ph. D., Editors. Fabrikoid, 1,294 pages. Price, $10.75. Philadelphia and Toronto, Blakiston Company, 1949.

In their advertisements, the publishers of this valuable addition to medical books have described the costs in time and money incident to its production, as well as the large staff and sources used in its compilation. Even a casual reference to its contents will convince anyone that these expenditures were worth while.

All editions are attractive as to type, spacing, and column arrangement. The thousands of definitions have evidently been agreed upon by those who use the words rather than being merely carry-over descriptions from older sources or

[1]*Stephen Weisz, M. D., Dallas.*
[2]*S. N. Key, Jr., M. D., Austin.*

from those not actively engaged in their use. A bolder, but just as useful, stroke has been to omit many words to be found nowhere except in other dictionaries. The hazard of mispronunciation is minimized by simpler keys and phonetic spelling.

The book contains 1,294 pages, of which the last 135 comprise the appendix. The latter contains seventeen sections consisting of tables or groups of words on certain topics, including anatomy, blood constituents, diets, the elements, enzymes, hormones, signs and symptoms, pathogenic organisms, anomalies and monsters, phobias, pregnancy, prescription writing, proteins, thermometric equivalents, veterinary doses, vitamins, and weights and measures.

The most gratefully received feature of such a volume should be the alphabetization of all terms.

Those in all medical fields will no doubt come to use and recognize the "New Gould Medical Dictionary" as the best available source of its type. A convincing casual test is to consult it for words which are appearing in the special journals and with which the reader is often unfamiliar. This reviewer found only one partial failure in forty trials.

[3]Handbook of Diseases of the Skin

Richard L. Sutton, M. D., Emeritus Professor of Dermatology and Syphilology, University of Kansas Medical School; and Richard L. Sutton, Jr., M. D., Associate Professor of Dermatology and Syphilology, University of Kansas Medical School, Cloth, 749 pages. Price, $12.50. St. Louis, C. V. Mosby Company, 1949.

This book is the most recent product of the prolific and skillful authorship of the Suttons, *père et fils.* (Actually, this book was written by Richard L. Sutton, Jr., who explains the cogent reasons why the senior authorship is accredited.)

Perusal of a Sutton book for the perceptive reader is a bit like a stroll through the monkey house at the zoo. One constantly encounters the unexpected. Dr. Sutton has his ideas; he presents them with a clear, usually forceful, sometimes pungent, but invariably dexterous and concise use of the English language. It should not be inferred, however, that this book is only a monograph or that it concerns itself solely with controversial material; the book actually touches upon the entire field of skin and allied disorders.

By following the policy of "mentioning almost everything" because the material has been presented and classified in a manner original with the Suttons, a useful book has been achieved. It is useful in a great many respects—practical for the average clinician, scholastic for him who desires to delve, but particularly useful for the reader who wishes to orient himself and attain dermatologic perspective rather quickly. The author pursues his own ideas on nosography; almost always his departures from the conventional operate toward clarification and sometimes toward debunking.

The illustrations are clear and intelligently chosen. An especially commendable feature is the handling of bibliographic references, the majority being incorporated directly into the text.

With all the features and virtues of this treatise, the distinctive characteristic to this reviewer is the fact that on any point of conceivable controversy, the author's opinion is clearly there, usually couched in unexpectedly piquant terms. Sometimes his wit, though subtle, is devastating to notions or practices in which he fails to find the justification of good sense.

There should be few medical bookshelves upon which this book would fail to find a useful spot.

[3]*Morris Polsky, M. D., Austin.*

[4]Medicine Throughout Antiquity

Benjamin Lee Gordon, M. D., Member American Association of the History of Medicine and American Academy of Ophthalmology and Otolaryngology. Cloth, 818 pages. Price, $6. Philadelphia, F. A. Davis Company, 1949.

Proof of the superiority of this work is the high praise given it by Dr. Max Neuburger, the historian, in his foreword. He says, "The author of this volume is one of the few practitioners who combines a lively interest in the history of medicine with sound and solid method in historical research."

The prehistoric background of ancient medicine is compiled in a way that is clear and readable. Medical beginnings in Babylon, Egypt, Persia, and China are described in separate chapters that assemble all the known facts of each period.

The contributions to medicine by the great nonmedical sages such as Herodotus, the archeologists like Henry Breasted and Flinders Petrie, and others are enumerated side by side with the better known works of Hippocrates, Galen, and the Arab physicians. This is an unusual book in its approach to medical history and its avoidance of the usual "re-hash" of known historical facts.

[5]A Synopsis of Physiology

A. Rendel Short, B.Sc., M. D., F.R.C.S., Late Professor of Surgery, University of Bristol, and Late Examiner Primary F.R.C.S.; C. L. G. Pratt, O.B.E., M.A., M.D., M.Sc., Lecturer in Mammalian Physiology, University of Cambridge, and Fellow of Christ's College, Cambridge; and C. C. N. Vass, M.Sc., Ph.D., M.B., Ch.B., Reader in Physiology in the University of London. Fourth Edition. Cloth, 346 pages. Price, $6. Baltimore, Williams & Wilkins Company, 1948.

This book fulfills well the authors' purpose to provide a summary of human physiology. It is written in outline form, systematically arranged to cover the various divisions of physiology, and contains an adequate number of charts and tables.

Synopses of this type, while apparently popular in England, have not found as much favor in this country. It does provide a rather complete review of physiology and an attempt is made to correlate physiologic observations and phenomena with clinical application. The chapters on carbohydrate and fat metabolism are marred by not containing information which has been derived from the use of radioactive isotopes.. The book could therefore not be recommended as really up to date. It might provide a quick review for use by candidates for the specialty board examinations. The index is complete, but no effort is made to provide a bibliography. On the whole, if one has need for a summary of physiology, this volume will be found satisfactory.

[6]Bacterial and Mycotic Infections of Man

Rene J. Dubos, Ph. D., The Rockefeller Institute for Medical Research, Editor. Cloth, 785 pages. Price, $5. Philadelphia, J. B. Lippincott Company, 1948.

This book is recommended to bacteriologists, health officers, public health nurses, and others as a source of knowledge of the bacteria, actinomycetes, and molds pathogenic for man, and the phenomena which characterize the infectious process.

The authors regard medical microbiology as a study of host-parasite relationships and not alone that of micro-

[4]*Walter Stuck, M. D., San Antonio.*
[5]*Ralph G. Greenlee, M. D., Temple.*
[6]*J. V. Irons, Ph. D., Austin.*

organisms considered as independent living agents. The presentation of the several pathogenic agents is such that extensive treatment of a few selected examples focuses attention on the underlying principles of infection, immunity, and allergy.

While this treatise is the result of a cooperative effort of many experts and consequently reflects their individual outlook, there is evidence of uniformity and coherence.

The price should not be a factor limiting the number of readers, since the National Foundation for Infantile Paralysis has subsidized the cost of preparation and publication of the book.

[7]Hematology

Willis M. Fowler, M. D., Professor of Internal Medicine, University of Iowa, Iowa City. Revised Second Edition. Cloth, 535 pages. Price, $8.50. New York, Paul B. Hoeber, Inc., 1949.

This textbook on hematology is clearly and concisely written. It is primarily for medical students and should serve its purpose well. This book does not improve or replace any of several other textbooks on hematology. The discussion of hemolytic anemias is especially good, but the discussion of hematology in infancy and childhood is rather brief.

[8]Diseases of the Ear, Nose and Throat

William Wallace Morrison, M. D., Professor of Otolaryngology and Attending Otolaryngologist, New York Polyclinic Medical School and Hospital; Senior Assistant Surgeon in Otolaryngology, New York Eye and Ear Infirmary; Associate Clinical Professor of Otolaryngology, New York University College of Medicine; Captain, Medical Corps (U.S.N.R.). Cloth, 772 pages. Price, $8.50. New York, Appleton-Century-Crofts, Inc., 1948.

This book shows careful planning and consideration for the reader, be he student or practitioner. It is well outlined and simply and ably presented to bring out the subject matter in a comprehensive manner. The subject discussed is well illustrated by clear, understandable drawings and diagrams. From the description given, the student can readily recognize his case in hand and get a comprehensive understanding of his problem.

The book, though having 772 pages, is not heavy. It is divided into eight sections. The first deals with introductory matters and explains the importance of a well-taken history and the necessary equipment used in the examination and treatment. The author presents an important discussion of the action of chemotherapeutic drugs. In the other seven sections he deals individually with each major classification of the field of otolaryngology. This reviewer particularly likes the way the author handles the functional test of the "ear" with suitable accompanying illustrations and diagrams. The other subjects are equally well handled as to their etiology, pathology, symptoms, diagnosis, and treatment. This book is well indexed and is heartily recommended. It is a good guide to have on one's desk for ready reference.

[9]Posttraumatic Epilepsy

A. Earl Walker, M. D., Professor of Neurological Surgery, The Johns Hopkins University, Baltimore. Cloth, 86 pages. Price, $2.75. Springfield, Ill., Charles C. Thomas, 1949.

This is an excellent and inclusive review of the present status of one of the most disconcerting problems that a physician encounters. Its inviting brevity and refreshing clarity make it both informative and enjoyable.

Its scope covers not only the clinical aspect and therapeutic possibilities, but also the chemical, physiologic, and neurologic changes in the brain that are being studied in search of a better understanding of the actual mechanism behind an epileptic seizure. Though these studies are inconclusive to date, the material contained herein should be encouraging to the practitioner and to the patient, and the presentation of this information should result in the referral of more of these cases to institutions where this work is performed.

This monograph might well serve as a blueprint in the management of posttraumatic epilepsy. No elaboration is made on the controversial points, and consequently, while the reader is well oriented by the material presented, he is not confused by various theories. Likewise, general acceptance of this treatise would do much toward removing most erroneous concepts of etiology with which the patient has previously been plagued. Psychotherapy as a therapeutic measure is stressed.

I would heartily recommend this monograph as a valuable addition to the library of any physician. It is well bound and its bold printing is a credit to the publisher.

[10]Geriatric Medicine

Edward J. Stieglitz, M. S., M. D., F.A.C.P., Editor; Attending Internist, Suburban Hospital, Bethesda, Md.; Doctor's Hospital, Washington, D. C.; Attending Internist (Geriatrics), Chestnut Lodge, Rockville, Md.; etc. Second edition. Cloth, 773 pages. Price, $12. Philadelphia and London, W. B. Saunders Company, 1949.

This book should be of interest to practically all physicians, and is a "must" for the general practitioner and internist. It consists of the contributions of a large number of distinguished authors, capably edited by Dr. Stieglitz, who also covers much of the general field of gerontology himself. Allowing that many of the causes and factors of aging are partially or totally obscure, there is still a great deal of information presented and a clear orientation toward the still unsolved problems of gerontology.

The portion of the book devoted to the prophylaxis and care of diseases peculiar to or common in the aging and aged is in general well devised and written. At times the authors are so limited as to space that the reader feels he is reading headlines. One may not entirely agree with everything in the book—notably, fasting for bleeding peptic ulcer, the value of calomel, and the homeopathic usage of vitamin K—but its good qualities so greatly outweigh any minor defects that it is strongly recommended as a permanent addition to any medical bookshelf.

[11]Transactions of the American Neurological Association

H. Houston Merritt, M. D., Secretary of the Association, Editor. Cloth, 247 pages. Richmond, Va., William Byrd Press, Inc., 1949.

The general arrangement of the transactions remains as in preceding years, consisting of lists of members, the presidential address, various papers presented at the 1948 session held at Atlantic City, N. J., and the minutes of the executive session.

Dr. George Wilson, the president, calls for a revival of interest in neurology, especially among younger men.

The contents of the various papers presented are highly technical and many are pure research. Of particular interest to the general practitioner would be the papers devoted to

[7]*George Clifford Thorne, M. D., Austin.*
[8]*Philip B. Greenberg, M. D., Beaumont.*
[9]*William R. Leon, M. D., Fort Worth.*
[10]*Wylie F. Creel, M. D., Austin.*
[11]*John L. Otto, M. D., Galveston.*

the study of headache, apoplexy, and chemotherapy of infections of the central nervous system.

Haymaker and Kernohan present the clinicopathologic findings in 50 cases of Landry-Guillain-Barré syndrome, and emphasize that the condition is a polyradiculoneuropathy affecting both sensory and motor components either singularly or together.

Surgical treatment of intracranial hemorrhage, that is, evacuation of the clot, in 14 cases as reported by Grant and Austin is of special interest. These authors recommend surgical intervention when the diagnosis and localization of the hemorrhage can be made.

Scarff says that unilateral prefrontal lobotomy for the relief of intractable pain is being done with the idea that less intellectual dulling will result.

Perhaps the most stimulating portion of the volume is the presentation of the discussion following each paper. In most instances it is lively and no punches are pulled. This volume is essential for those who are interested in what's new in neurology.

[12]Photoradiography in Search of Tuberculosis

David Zacks, M. D., Chief of Clinics, Massachusetts Department of Public Health. Cloth, 297 pages. Price, $5. Baltimore, Williams & Wilkins Company, 1949.

A chest roentgenogram for every person is offered as the immediate objective in mass chest radiography. In order to bring this about, education regarding pulmonary tuberculosis is necessary, not only to the patient but also to the officials and voluntary health agency workers. There are many problems the critical appraiser of mass roentgen-ray methods should consider, such as isolation, treatment, supervision, socio-economics, and psychology.

This book deals with the organization, personnel, operation of mobile units, and the planning and execution of mass chest roentgen-ray service. Described clearly and simply are the many details essential for planning this type of survey. An atlas of pulmonary lesions, including artefacts, and a chapter on minimal pulmonary tuberculosis are presented. The closing chapters relate to education and correspondence. The program for high school study includes questions and answers. This is a new and acceptable approach to the education of the public to periodic chest examinations.

This small volume covers considerable ground in bringing to the physician, public health worker, and business enterprise the fundamentals of mass photoradiography.

[12]*Ben DuBilier, M. D., Austin.*

[13]Shearer's Manual of Human Dissection

Charles E. Tobin, Ph. D., Associate Professor of Anatomy, University of Rochester School of Medicine and Dentistry, Editor. Second edition. Cloth, 286 pages. Price, $4.50. Philadelphia and Toronto, Blakiston Company, 1949.

To do a book of this sort justice, one would have to admit that the modern method of teaching anatomy is desirable. It was written, it seems, to meet the student's desire for a simple guide in dissection. The author seems to be apologizing for its publication.

While the brief descriptions given of structures are accurate as far as they go, they are too brief to be of much value in obtaining a concise picture of the anatomic structures described. It would appear that additional space could have been devoted to giving more detailed dissection directions, leaving it to the student to obtain the descriptive material from more adequate texts.

The author has done well considering the purpose for which the book was written, but it is regrettable that such a purpose exists. The book can be of little value to the practicing physician.

[13]*W. W. Looney, M. D., Dallas.*

ORGANIZATION SECTION

STATE MEDICAL ASSOCIATION

LOCAL COMMITTEES PREPARE FOR ANNUAL SESSION

Membership of the committees from Tarrant County Medical Society which will be responsible for arrangements for the 1950 annual session of the State Medical Association have been announced recently by Dr. T. H. Thomason, chairman of the Committee on General Arrangements. These committees are available to assist physicians throughout the state who have problems concerning the annual session, and inquiries may be addressed directly to the chairman of the appropriate committee.

Serving with Dr. Thomason on the Committee on General Arrangements are Drs. May Owen, Tom B. Bond, W. P. Higgins, Jr., and W. F. Parsons. The other committees are as follows:

LOCAL COMMITTEES

Hotels.—W. F. Parsons, Chairman; A. W. Butler, Jr., J. T. Tucker, Jr.

Information.—Charles H. McCollum, Jr., Chairman; J. Franklin Campbell, Charles J. Terrell, William W. Wood, Jr.

Finance.—W. P. Higgins, Jr., Chairman; James A. Hallmark, Frank G. Sanders.

Memorial.—L. H. Reeves, Chairman; A. D. Ladd, J. T. Tucker.

Clinical Luncheons.—Frank Schoonover, Jr., Chairman; W. S. Barcus, James O. Chambers, C. C. Garrett, R. L. Grogan, Frank W. Halpin, William W. McKinney, James W. Short, W. B. Swift.

Alumni and Fraternity Banquets.—Henry Grammer, Chairman; J. W. Tottenham, Jr. (Texas), George W. Lacy (Tulane), C. O. Terrell, Jr. (Baylor), William W. McKinney (Alpha Kappa Kappa), Mal Rumph (Nu Sigma Nu), J. R. Wise (Phi Beta Pi), George Y. Siddons (Phi Chi), A. I. Goldberg (Phi Delta Epsilon), A. W. Butler, Jr. (Theta Kappa Psi), J. D. Murphy (Phi Rho Sigma).

Reception.—Theron H. Funk, Chairman; M. H. Crabb, William M. Crawford, O. P. Griffin, Hub E. Isaacks, W. S. Lorimer, Jr., S. J. R. Murchison.

Transportation.—Oscar W. Haafke, Chairman; L. N. Gilliland, Jr., George W. Lacy, Johnnie E. Monaghan.

Scientific Exhibits.—John M. Church, Chairman; John J. Andujar, R. G. Lemon.

Technical Exhibits.—Ernest E. Anthony, Jr., Chairman; Tilden L. Childs, Jr., J. C. Tom, Jr.

Halls and Lanterns.—DeWitt Claunch, Chairman; Frank Cohen, D. E. Neal.

Publicity.—Hobart O. Deaton, Chairman; Robert D. Bickel, Ray V. Brasher.

Women Physicians.—Elizabeth A. Taylor, Chairman; Anna M. Greve, Blanche Terrell.

Golf.—H. C. Thomas, Chairman; Dolphus E. Compere, Frank J. Daugherty, R. H. Gough, F. L. Snyder.

Skeet Shooting.—James H. Hook, Chairman; Clive R. Johnson, Val D. Scroggie.

While the local committees are completing arrangements in Fort Worth for the May 2-4 meeting, a number of statewide committees with responsibilities for the annual session are also at work. Among these is the Council on Scientific Work, which will meet in mid-January to close the scientific program. Any member of the Association who has a paper which he would like to submit for consideration by one of the nine sections should send it to the appropraite section officers immediately. The names of these officers are listed in the August issue of the JOURNAL, page 601.

COUNTY SOCIETIES

Bell County Society

October 5, 1949

(Reported by P. M. Ramey, Secretary)

General Principles of Parenteral Fluids from Clinician's Standpoint—Carl A. Moyer, Dallas.

Bell County Medical Society met in Temple on October 5 with 105 members and guests present. Herman Rice, Ray B. Greenbill, and L. L. Thompson, Temple, were accepted into the society.

R. D. Moreton moved that a committee be appointed with W. N. Powell as chairman to continue the work of the diabetes detection program. The motion was seconded and approved.

Carl A. Moyer, Dallas, spoke on the topic given above.

Brazoria County Society

October 27, 1949

(Reported by W. D. Nicholson, Secretary)

Interpretation of Roentgenograms Taken During Tuberculosis Survey—Howard E. Smith, Austin.

Members of Brazoria County Medical Society met October 27 in West Columbia. Howard E. Smith, head of the Tuberculosis Division of the State Health Department, was the speaker.

C. E. Fuste, Jr., Alvin, reported that the application of the society for a charter has been approved by the Board of Councilors. W. C. Holt, Angleton, was appointed by the president to fill out the questionnaire which the state has requested in a survey to determine the number of beds needed for hospitalization in the area.

Brazos-Robertson Counties Society

September 20, 1949

Surgical Conditions of Esophagus—G. V. Brindley, Jr., Temple.

At the September 20 meeting of Brazos-Robertson Counties Medical Society in Bryan, G. V. Brindley, Jr., Temple, was the guest speaker.

Cooke County Society

October 11, 1949

Problems in Urology—Paul W. Matthews, Dallas.

Members of Cooke County Medical Society had as their guests dentists of the county at the October 11 meeting in Gainesville. Dr. and Mrs. Ira L. Thomas served dinner in their home, where the meeting was held.

Paul W. Matthews, Dallas, spoke on the subject given above.

Cherokee County Society

September 27, 1949

Medicine Against Compulsory Health Insurance—George A. Schenewerk, Dallas.

George A. Schenewerk, Dallas, president of Dallas County Medical Society, spoke to about fifty members of the Cherokee County Medical Society and Auxiliary at a dinner meeting September 27 in Jacksonville.

After the program auxiliary members held a brief business session at which Mrs. C. L. Jackson, Rusk, president, presided.

Dallas County Society

October 27, 1949

(Reported by W. W. Fowler, Secretary)

Symposium on Radioactive Materials:
- Production of Radioactive Materials Used in Medicine—Allen Reid, Ph. D., Dallas.
- Application of Radioactive Materials in Clinical Medicine—Julian Acker, Dallas.
- Health Hazards from Radioactive Elements—Henry Lanz.
- Radioactive Isotopes as a Form of Medical Therapy—J. R. Maxfield, Jr., Dallas.

Dallas County Medical Society met October 27 in Dallas with the vice-president, Edwin L. Rippy, presiding. George Jones urged members to contribute to the Community Chest.

The symposium outlined above was presented.

November 10, 1949

(Reported by W. W. Fowler, Secretary)

Developmental Vestiges and Their Pathology—Rupert A. Willis, London, England.

George A. Schenewerk, president, presided at the business session of the Dallas County Medical Society in Dallas on November 10.

Wayne Hanson Granger and Irvin Deutsch were accepted as members by transfer. Joseph M. Hill invited members to attend a meeting of the Texas Blood Bank Association in Dallas in December.

Ben Merrick, program chairman, presented John L. Goforth, who introduced the speaker, Rupert A. Willis, pathologist of London, England.

Gonzales County Society

October 12, 1949

(Reported by David M. Shelby, Secretary)

Appendicitis in Childhood (motion picture).
Review of Anticoagulant Therapy—James C. Price, Gonzales.

Gonzales County Medical Society held a dinner meeting October 12 in Gonzales with 100 per cent attendance. Duane Schram, chief of physical medicine and rehabilitation of Gonzales Warm Springs Hospital for Crippled Children, and Mrs. Schram were guests.

The program outlined above was given. Mr. Lee Muenzler, instructor in science and visual education at the Gonzales High School, served as projectionist.

Gray-Wheeler-Hansford-Hemphill-Lipscomb-Roberts-Ochiltree-Hutchinson-Carson Counties Society

October 18, 1949

(Reported by H. L. Wilder, Secretary)

Streptomycin in Tuberculosis—John W. Middleton, Galveston.
Discussion—J. B. White, Amarillo.
Case Reports: Reduplication of Ureter (roentgenograms)—D. P. Bonner, Pampa.

A dinner meeting was held by Gray-Wheeler-Hansford-

Hemphill - Lipscomb - Roberts - Ochiltree - Hutchinson - Carson Counties Medical Society in Pampa on October 18. Twenty-one members and four guests were present.

The program outlined above was presented. Dr. Middleton stressed that streptomycin can produce resistant stains and that while it is a valuable remedy it should not be given alone but with other recognized forms of treatment.

Hardeman-Cottle-Foard-Motley Counties Society

September 15, 1949

Seventeen members attended the September 15 meeting of Hardeman-Cottle-Foard-Motley Counties Medical Society in Matador. A banquet was followed by addresses and discussions.

Kerr-Kendall-Gillespie-Bandera Counties Society

October 10, 1949

(Reported by Roger Stevenson, Secretary)

Kerr-Kendall-Gillespie-Bandera Counties Medical Society met October 10 in Kerrville. Sixteen members were present.

Mr. Philip Overton, Austin, general attorney for the State Medical Association, reported on the progress of the state and national campaign against socialized medicine. Also present at the meeting was Representative Fred Niemann, Yoakum, a member of the State Legislature.

November 14, 1949

(Reported by Roger Stevenson, Secretary)

Management of the Gynecologic Patient—John D. Weaver, Austin.

The monthly meeting of Kerr-Kendall-Gillespie-Bandera Counties Medical Society was held in Fredericksburg on November 14. After the business session, John D. Weaver, Austin, talked on the above subject. He divided the management of the gynecologic patient into several categories and recommended that the physician place his patient in one of these categories and then decide how to treat her.

Lamar County Society

November 3, 1949

(Reported by T. E. Hunt, Jr., Secretary)

The Lamar County Medical Society met in Paris on November 3 with fourteen members and two visitors, R. T. Payne and C. B. Reed, both of Clarksville, present.

H. W. Parchman, president, presided. M. A. Walker, Jr., Paris, reported on the September meeting of the Texas Association of Obstetricians and Gynecologists. D. R. Lewis, Paris, gave an account of his recent visit to leading medical centers in the eastern states.

Lubbock-Crosby Counties Society

November 1, 1949

(Reported by M. D. Watkins, Secretary)

The Lubbock-Crosby Counties Medical Society met November 1 in Lubbock.

Miss Mary Lou Hawkins, child welfare supervisor in Lubbock for the State Department of Public Welfare, explained the child welfare program carried on in Lubbock and throughout the state. Richard K. O'Loughlin and Robert W. Moore were elected to membership in the society.

Ivan Mayfield reported that during the Diabetes Detection Drive, 1,407 urinary specimens were tested in Lubbock and that 21 cases of glycosuria were detected. He said that the City Health Department had sent letters to the physicians of those persons found to have glycosuria.

Albert G. Barsh moved and Byron A. Jenkins seconded that the tuberculosis survey to be made by the roentgen-ray mobile unit in Crosby County receive the approval of the society. The motion carried. It was understood that the survey while having the sanction of the society would be requested by the physicians of Crosby County.

Frederick P. Kallina reported that there is only one laboratory in Lubbock which is recognized to perform serologic tests for premarital and prenatal blood examinations. He explained the different forms to be used and methods of securing and sending blood to the laboratory, and distributed sample forms to the members.

Nacogdoches County Society

October 12, 1949

Chest Lesions (roentgenograms)—Robert Taylor, Lufkin.

Nacogdoches County Medical Society met October 12 in Nacogdoches for a discussion of chest lesions and tuberculosis.

Plans for the formation of a tuberculosis association in Nacogdoches County were discussed. James G. Taylor was instructed to inform the Nacogdoches County Junior Chamber of Commerce of the willingness of the medical society to cooperate fully in forming the tuberculosis unit. Arrangements were discussed to bring a mobile roentgen-ray unit to Nacogdoches to make chest examinations.

Potter County Society

October 10, 1949

Robert Shaw and Elliott Mendenhall, chest specialists of Dallas, were guest speakers at the October 10 meeting in Amarillo of Potter County Medical Society. A dinner party was given before the meeting by the president, J. B. White, Amarillo.

Smith County Society

October 13, 1949

Diagnosis of Liver Diseases (slides)—John S. Bagwell, Dallas.

The first fall meeting of Smith County Medical Society was held October 13 in Tyler. Forty-three members were present.

Col. T. J. Cassidy, representing the Red Cross, asked the cooperation of the society in forming a blood bank. The society voted its approval and a committee consisting of Mullowney Anderson and Jesse Goldfeder was appointed to work with the Red Cross.

Resolutions on the death of Dr. J. R. Dickson, Arp, were read. David B. Connery, H. W. Poetter, and George W. Burch were accepted into membership by transfer.

James M. Vaughn, Lex Neill, and Dr. Goldfeder were appointed to confer with city and county officials in regard to matters pertaining to public health.

John S. Bagwell, Dallas, spoke on the topic given above.

Tarrant County Society

October 18, 1949

(Reported by W. P. Higgins, Jr., Secretary)

Rupture of Gallbladder—W. P. Higgins, Jr., Fort Worth.
Discussion—F. L. Snyder, Burgess Sealy, J. D. Murphy, William M. Crawford, and T. H. Thomason, Fort Worth.
Congenital Anomalies of Digestive Tract of Surgical Interest in Infants and Children—R. J. White, Fort Worth.
Discussion—Frank Cohen, Burgess Sealy, R. G. Lemon, F. L. Snyder, and Hobart O. Deaton, Fort Worth.

At the October 18 meeting of Tarrant County Medical Society in Fort Worth the program outlined above was given. Sixty-six members and three visitors were present.

N. A. Bryan was elected to membership. M. H. Crabb announced that Armour and Company had made available free to doctors eighty barrels of intravenous sets.

May Owen made an announcement concerning the Southwest Regional Cancer Conference on November 9 and the combined tumor clinic to be held the following day.

November 1, 1949

(Reported by W. P. Higgins, Jr., Secretary)

Preservation of Facial Nerve in Parotid Gland Tumors—William O. Ott, Fort Worth.
Discussion—R. J. White and T. H. Thomason, Fort Worth.
Ureterosigmoid Transplant Employing Combined Mucosa-to-Mucosa and Pull-Through Technique—Keller Doss, Fort Worth.
Discussion—D. E. Compere and Hub E. Isaacks, Fort Worth.

Sixty-one members and three visitors attended the November 1 meeting of Tarrant County Medical Society in Fort Worth. The program was given as outlined above.

The following were elected to membership on application: William M. Branch, Jr., Frederick C. Rehfeldt, and Thomas U. Taylor, II. Members accepted on transfer were John B. Patterson and David E. Small.

Tribute was paid to Dr. Wilmer Allison, who died October 28.

T. C. Terrell stated that a representative from the federal Bureau of Narcotics had called his attention to the widespread negligence of physicians in conforming with the requirements of the Harrison Act. He urged members to join the American Association of Physicians and Surgeons in its fight against socialized medicine.

Tom Green-Eight County Society

October 24, 1949

(Reported by M. D. Knight, Secretary)

Thirty-five members attended the special meeting of Tom Green-Eight County Medical Society on October 24 in San Angelo. Guest speaker was Mr. Philip Overton, Austin, general attorney for the State Medical Association.

The public relations activities of state and national medical organizations were explained by Mr. Overton. He also mentioned several bills of significance to health which were passed at the recent session of the State Legislature and the status of national legislation concerning health.

The society was informed by Mr. Overton that its contribution to the American Medical Association assessment is only 38 per cent and that Texas ranks forty-third in contributions. He asked for cooperation in this regard and suggested that individual letters be sent to delinquent members.

DISTRICT SOCIETIES

Eleventh District Society

October 26, 1949

About fifty doctors attended the annual fall meeting of the Eleventh District Medical Society in Jacksonville on October 26. Lynn Hilbun, president, was the presiding officer.

G. V. Brindley, Temple, president of the State Medical Association, was the honor guest at the noon luncheon. Officers elected at the meeting were Edwin G. Faber, Tyler, president, and John Travis, Jacksonville, secretary.

Fifteenth District Society

October 25, 1949

Welcome Address—Mr. R. B. Williams, Mayor of Longview.
Treatment of Renal and Ureteral Calculi—J. W. Dworin, Longview.
Acute Upper Respiratory Infections in Infants and Children—Clarence H. Webb, Shreveport, La.
Nutrition—Joe Nichols, Atlanta.
Endocrine Gynecology—Karl John Karnaky, Houston.
Treatment of Inguinal Hernia in Infants and Children—J. W. Duckett, Dallas.
How to Take Care of the Nervous Patient in the Office—Stephen Weisz, Dallas.
Poliomyelitis: Recent Advances—D. K. Barnes, Dallas.
Diagnosis and Treatment of Coronary Artery Disease—Charles N. LaDue, Dallas.

The Fifteenth District Medical Society met in Longview on October 25 and the program outlined above was given. Forty-eight doctors attended.

A business meeting was held, and at the luncheon G. V. Brindley, Temple, President of the State Medical Association of Texas, was guest speaker. Joe Nichols, district councilor, also gave a report. R. E. Slade, secretary of Gregg County Medical Society, was in charge of arrangements.

Officers elected were F. V. Mondrik, Marshall, president; J. L. Finlaw, Gilmer, vice-president; and Hardy Cook, Longview, secretary-treasurer. Joe Nichols, Atlanta, district councilor, was nominated to succeed himself.

AUXILIARY SECTION

AUXILIARY NEWS

Bexar County Auxiliary

The Woman's Auxiliary to the Bexar County Medical Society met in San Antonio on November 11. Miss Otela Kelley, guidance director at Jefferson High School, addressed the group. A brunch was served after the business meeting. Hostesses were Mrs. J. J. Martinez and Mrs. Belvin Pritchett.—Mrs. Dudley Jackson, Jr., Publicity Chairman.

Officers of the Woman's Auxiliary to the State Medical Association of Texas: President, Mrs. Joseph B. Foster, Houston; President-Elect, Mrs. William M. Gambrell, Austin; First Vice-President (Organization), Mrs. Paul Brindley, Galveston; Second Vice-President (Physical Examination), Mrs. Howard E. Puckett, Amarillo; Third Vice-President (Hygeia), Mrs. P. M. Kuykendall, Ranger; Fourth Vice-President (Program), Mrs. L. S. Thompson, Dallas; Corresponding Secretary, Mrs. Mark H. Latimer, Houston; Recording Secretary, Mrs. R. Ernest Clark, Memphis; Treasurer, Mrs. V. M. Longmire, Temple; Publicity Secretary, Mrs. R. T. Wilson, Austin; Parliamentarian, Mrs. Fred Sutton, Beaumont.

Cass-Marion Counties Auxiliary

Members of Cass-Marion Medical Society and Auxiliary held a dinner in Linden on October 13 and met separately after the dinner. Mrs. W. S. Terry, president, presided at the business session of the auxiliary held in the home of Mrs. C. E. Davies.

Cass-Marion Counties Auxiliary was entertained by the president, Mrs. W. S. Terry, at a dinner in her home in Jefferson on November 9. During the business session which followed, a report on the Fifteenth District Auxiliary meeting in Longview on October 25 was given.—Mrs. M. J. Brooks, Jr.

Cooke County Auxiliary

Members of Cooke County Auxiliary made plans at a luncheon in Gainesville on October 11 to hold a crippled children's clinic October 25. Two orthopedists, one from Dallas and one from Fort Worth, were to examine free of charge any crippled child and make recommendations. Mrs. J. A. Atchison was named chairman of a committee in charge of arrangements.

At the luncheon meeting a report was given concerning the well child conferences which the auxiliary sponsors. Two new members, Mesdames M. L. Monroe and J. McKay, were welcomed into the auxiliary. Fourteen members were present.

Dallas County Auxiliary

New members of the Dallas County Auxiliary were entertained at a tea October 28 at the home of Mrs. James B. Howell, Dallas. Mrs. Ben Harrison and Mrs. Howell with the sixteen new members received the guests.

Mrs. Eugene L. Aten was decorations chairman. Gold and bronze flowers in a copper bowl were used as the table centerpiece. Mrs. Gordon B. McFarland, president, and Mrs. Shirley Hodges, president-elect, poured coffee from brass samovars. Assisting in the dining room were members of the courtesy committee, Mrs. Leon Hodge, chairman, and the entertainment committee, Mrs. J. B. Bourland, chairman.

Included in the house party were Mesdames Don L. Paulson, L. B. Hurt, Jack Womack, Thomas Barnes, Joe Moody, E. G. Grafton, Fred Horn, and Lee J. Alexander.

The Woman's Auxiliary to the Dallas County Medical Society heard Dr. Edwin L. Rippy, vice-president of the society, speak on diabetes control in Dallas on November 2.

The executive board met the day preceding the auxiliary meeting in the home of Mrs. Marvin P. Knight. Other hostesses were Mesdames L. A. Mewhinney, Hudson Dunlap, Manning Shannon, and F. E. Gessner.—Mrs. M. P. Knight, Publicity Chairman.

El Paso County Auxiliary

A luncheon on October 10 in El Paso was the first meeting of the season of the Woman's Auxiliary to the El Paso County Medical Society.

Mrs. J. Leighton Green, incoming president, presided at the business meeting after the luncheon. A one-act play, "Rehearsal For Duty," written by Mrs. H. D. Hatfield, ways and means chairman, was given. The play depicted the responsibilities and obligations of each officer, committee chairman, and member of the auxiliary. Mrs. James J. Gorman was the narrator. Members of the cast were Mesdames George Turner, A. D. Long, Harry H. Varner, J. Leighton Green, Louis Breck, Delphin von Briesen, Newton Walker, Joe C. Carter, Maynard S. Hart, Clement C. Boehler, Leigh S. Wilcox, John Martin, and Arlin B. Cooper.

Mesdames Celso C. Stapp, Vincent M. Ravel, and Edmund P. Jones were in charge of the luncheon and were assisted by Mesdames B. F. Stevens, Jacob Rogde, William J. Davis, Harold Edinoff, L. C. Feener, B. Lynn Goodloe, J. Houston Hinton, John H. Johnstone, Sam R. King, Martin H. Leonard, Z. Ramon Molinar, P. R. Outlaw, Richard J. Phillips, William M. Tubbs, M. R. Vinikoff, W. W. Woolmann, Wanda Deter, and Leopoldo Villareal.

Kerr-Kendall-Gillespie-Bandera Counties Auxiliary

Mrs. Joseph B. Foster, Houston, President of the Woman's Auxiliary to the State Medical Association of Texas, and Mrs. E. L. Dyer, Kerrville, president of the county auxiliary, were honored at a tea by Kerr-Kendall-Gillespie-Bandera Counties Auxiliary in the home of Mrs. Sam Thompson, Kerrville, on October 17. Other guests were Mrs. W. S. Red, Houston, and Mrs. R. C. Bellamy, Daisetta.

Mrs. Foster outlined the year's work for the State Auxiliary and gave the standing of various state auxiliaries in activities carried on throughout the nation. Mrs. A. P. Allison, Kerrville, reported on the health program which will be held in cooperation with the teachers in the public schools. Mrs. W. E. Bell, Kerrville, Fifth District council woman, reported on the recent meeting in San Antonio.

Liberty-Chambers Counties Auxiliary

A business meeting of Liberty-Chambers Counties Auxiliary was held in the home of Mrs. D. H. Davies, Liberty, on October 13. Mrs. D. P. Schulz, president, presided, and six members were present. Refreshments were served.—Mrs. A. L. Delaney, Secretary.

Nacogdoches County Auxiliary

An annual benefit tea was given by Nacogdoches County Auxiliary on November 16 in the home of Mrs. J. Frank Beall, Nacogdoches. Proceeds from the tea will help furnish the new hospital.

Mrs. James G. Taylor, Jr., president, presided at a business meeting of the auxiliary earlier in the month. Twelve members were present and Mrs. E. P. McKinney was hostess.

Rusk-Panola Counties Auxiliary

The Woman's Auxiliary to the Rusk and Panola Counties Medical Societies held its monthly meeting October 18 in Carthage at the home of Mrs. H. D. Kuykendall. Mrs. J. C. Allen, Henderson, president, presided. Fifteen members and two guests, Mrs. C. E. Willingham and Mrs. P. M. Bailes, both of Tyler, were present.

The new yearbook and the program for the coming year were the chief topics discussed. Before the meeting the auxiliary had dinner with their husbands and heard Dr. C. E. Willingham, Tyler, Eleventh District councilor, speak on "Medical Ethics."—Mrs. K. C. Prince, Reporter.

Smith County Auxiliary

A coffee in October opened the season's activities for Smith County Auxiliary. Co-hostesses for the coffee, given in the home of Mrs. Jesse Goldfeder, Tyler, were Mesdames Goldfeder, Hascall Muntz, and Ben Wilson. Mrs. E. C. Clawater presided at the coffee service.

After the coffee Mrs. Glynne Brown, president, presided during a short business session. Mrs. E. D. Rice read the auxiliary's creed. Mrs. Lex Neill, yearbook chairman, distributed yearbooks.

Mrs. Neill and Mrs. R. E. G. Baldwin outlined the auxiliary's project for the year. The auxiliary will sponsor a "Career Week" for girls in their junior and senior year in high school and for girls in junior college. While various careers will be included, emphasis will be placed upon nurse recruitment, and guests from Baylor University School of Nursing will explain the program.

Mrs. Henry Poetter and Mrs. William D. Burch were welcomed as new members.

Smith County Auxiliary members attended a coffee meeting at the home of Mrs. Thomas M. Jarmon, Tyler, early in November. Co-hostesses were Mrs. E. H. Caldwell and Mrs. R. E. G. Baldwin.

Ben Johnson, local attorney, spoke on the Gilmer-Aikin plan. A business session followed his talk. Thirty members attended, and Mrs. R. T. Travis, Jacksonville, president of the Eleventh District Auxiliary, and Mrs. J. M. Travis, also of Jacksonville, were guests.

Autumn flower arrangements were placed in the reception rooms and formed the centerpiece on the table. Mrs. Ben Wilson presided at the coffee service.—Mrs. C. E. Willingham.

Tom Green-Eight County Auxiliary

In conjunction with the meeting of the Fourth District Auxiliary, the Tom Green-Eight County Auxiliary held a banquet for members of the county society and auxiliary November 6 in San Angelo. Hostesses were Mesdames Frederick Simpson, Carl Kunath, Joe L. Cornelison, J. W. Singleton, and W. E. Schulkey.—Mrs. W. Grady Mitchell, Publicity Secretary.

Fourth District Auxiliary

The Woman's Auxiliary to the Fourth District Medical Society met for luncheon in San Angelo on November 6. Mrs. W. B. Butner, San Angelo, district council woman, served as mistress of ceremonies.

No election of officers was held since Tom Green-Eight County Auxiliary is the only currently active unit in the district. Mrs. Butner pointed out the need for other auxiliaries in the district and offered assistance to groups wishing to organize. Mrs. Butner also stressed the need for members-at-large from localities which do not have auxiliaries.

After the business session Mrs. Butner introduced Mrs. J. C. Sturges, Jr., who reviewed "The Doctor Wears Three Faces" by Mary Bard.

Entertainment also included bridge and a tour of the city.—Mrs. W. Grady Mitchell.

Fifteenth District Auxiliary

The Fifteenth District Auxiliary met in Longview on October 25 in conjunction with the district medical society meeting. New officers elected include Mrs. J. H. Harris, Marshall, president; Mrs. E. L. Fender, Mount Pleasant, secretary-treasurer; and Mrs. J. E. Ball, Mount Pleasant, district council woman.

DEATHS

D. R. AVES

Dr. Delano Richard Aves, La Porte, Texas, died in a Houston hospital October 27, 1949, of enlargement of the heart and acute cardiac failure.

Born in Cleveland, Ohio, on November 5, 1887, Dr. Aves was the son of the Right Rev. Henry D. Aves, former Episcopal Bishop of Mexico, and Mary Gertrude (Smith) Aves. Receiving his preliminary education at Welch Academy, Houston, and Kenyon College, Gambier, Ohio, he was graduated from the University of Texas School of Medicine, Galveston, in 1913 and served an internship at Shumpert Memorial Hospital, Shreveport, La., from 1913 to

DR. DELANO R. AVES

1914. Dr. Aves was in charge of a small hospital in Mexico from 1914 until 1917. From 1917 to 1919 he served in the U. S. Army Medical Corps, first in France and then in New York. After his discharge as a captain in 1919, he was resident physician at Sacred Heart Sanatorium, Milwaukee, from 1920 to 1922. He practiced in Birnamwood, Wis., until 1928, when he moved to La Porte, practicing there until his death.

Dr. Aves was a member of the State Medical Association and the American Medical Association through Harris County Medical Society. He held a reserve commission of major in the U. S. Army Medical Corps from 1919 until 1939, and during World War II was a captain in the Texas Defense Guard. He held the rank of captain in the Texas National Guard at the time of his death. He was a member of the American Legion, the La Porte Chamber of Commerce, the Masonic Order, and the Episcopal Church. Last January Dr. and Mrs. Aves were voted the outstanding couple of the Bayshore area in a popular poll conducted by the La Porte Chamber of Commerce.

Dr. Aves is survived by his wife, the former Miss Laura Marion Mason, whom he married April 24, 1922, in Milwaukee; three daughters, Miss Priscilla Gertrude Aves, Dallas; Miss Mary Susan Aves, Iola, Kan.; and Miss Laura Mason Aves, La Porte; a sister, Mrs. U. H. Berthier, Monterrey, Mexico; and a brother, William Leonard Aves, Esmeralda, Coahuilla, Mexico.

H. C. ECKHARDT

Dr. Herman Charles Eckhardt, Yorktown, Texas, died at his home August 5, 1949, after a heart attack.

The son of Herman P. and Pauline (Heissig) Eckhardt, Dr. Eckhardt was born August 11, 1878, at Yorktown. He attended the local public school and Ford's Preparatory School, Austin, and received a degree in dentistry from Western Dental College, Kansas City, in 1901. He practiced dentistry in Yorktown until 1903, at which time he entered the University of Tennessee Medical Department, Nashville, being graduated in 1907. Dr. Eckhardt was an honor student throughout his school days and was awarded the faculty medal in medical school. He practiced medicine for short periods in Yorktown, Yoakum, Nordheim, and Gonzales until 1910, when he returned to Yorktown to practice until his death.

A member of the American Medical Association and the State Medical Association throughout his professional life, Dr. Eckhardt served as councilor of the Eighth District from 1935 to 1941 and as secretary of DeWitt County Medical Society from 1933 to 1935 and again in 1940, 1944, and 1948. He also represented the county society in the House of Delegates of the State Medical Association for many years.

On September 17, 1900, in Yoakum, Dr. Eckhardt married Miss Laura Hudson, who survives. Other survivors are two daughters, Mrs. F. M. Koch, San Antonio, and Mrs. Willard Moody, Yorktown; two sons, H. H. Eckhardt and

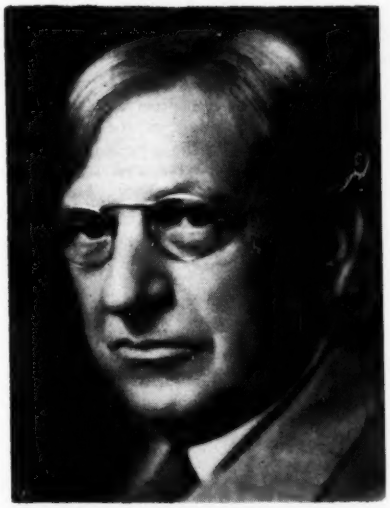
Howard Paul Eckhardt, both of Yorktown; seven grandchildren; and two great-grandchildren.

S. E. MILLIKEN

Dr. Samuel Edwin Milliken, Dallas, Texas, died September 18, 1949, at his home.

Dr. Milliken was born December 2, 1866, in Mansfield. Receiving his preliminary education in Thorp Spring, he was graduated from the University of Louisville Medical Department, Louisville, Ky., in March, 1887. He practiced in New York from 1889 to 1897, and in Dallas from that time until his retirement in 1941.

Throughout his professional career in Texas, Dr. Milliken was a member of the American Medical Association and the State Medical Association through Dallas County Medical Society, of which he was president in 1916. He was elected



DR. S. E. MILLIKEN

to honorary membership in the State Medical Association in 1946 and was chairman of the Section on Gynecology and Obstetrics in 1922. A fellow of the American College of Surgeons, Dr. Milliken was local surgeon for the Texas and Pacific Lines and the St. Louis Southwestern Railway Lines and was professor of surgery at Baylor University College of Medicine when it was located in Dallas. He was a member of the Masonic Order, Knights Templar, and Hella Temple Shrine and belonged to the Dallas Athletic Club, Lakewood Country Club, and High Noon Club. Dr. Milliken was a member of the Presbyterian Church.

Dr. Milliken is survived by his wife, Mrs. Kate Milliken, their daughter, Mrs. Katherine Busch, St. Louis, Mo.; a sister, Mrs. Maynie Griffin, Chickasha, Okla.; and four grandchildren.

L. D. RICHARDS

Dr. Lawrence Duval Richards, Seminole, Texas, died in a Lubbock hospital October 2, 1949, from a heart attack.

Born in Collin County, Texas, on October 7, 1875, he attended Farmersville High School. He attended the University of Tennessee and was graduated from Memphis Hospital Medical College, Memphis, in 1902. He began his practice in Celeste, moving to Paducah in 1903. Shortly afterward he moved to Plainview, where he and the late Dr. J. V. Guyton opened the first hospital in that city. In 1914 he went to Seminole, where he practiced until his death.

Throughout his professional career, Dr. Richards was a member of the American Medical Association and the State Medical Association, first through Hardeman-Cottle and then through Dawson-Lynn-Terry-Gaines-Yoakum Counties Medical Society; he was president of the latter society in

DR. LAWRENCE D. RICHARDS

1941. He was elected an honorary member of the State Medical Association in 1949.

Dr. Richards is survived by his wife, Mrs. Lucille Richards; two daughters, Mrs. Elliott M. Miller, Houston, and Mrs. Rosina Weider, Dallas; two step-daughters, Mrs. John D. Worthington, Pasadena, Calif., and Mrs. W. D. Hart, Jr., Seminole; four step-sons, R. L. Jones, Jim Jones, Horace Jones, and Harold Jones, all of Seminole; two brothers, W. R. Richards, Celeste, and J. D. Richards, Paducah; four sisters, Mrs. Nora Goodman, Mrs. Myrtle Havens, Miss Willie Richards, and Miss Ina Richards, all of Paducah; and one grandson.

M. J. TAYLOR

Dr. Martin Junius Taylor, Houston, Texas, died October 21, 1949, at his home from cardiac decompensation.

The son of Byron Ludwell and Charlotte (King) Taylor, Dr. Taylor was born September 28, 1870, in Marlin. He received his preliminary education in Moscow Masonic High School, Moscow, Texas, and was graduated in 1892 from the Memphis Hospital Medical College, Memphis, now the University of Tennessee College of Medicine. Dr. Taylor began his private practice in Moscow in 1892. Two years later he entered general practice and was surgeon for the W. T. Carter Brothers Lumber Company, moving to Camden, Texas, in 1897. In 1918 he moved to Houston, where he practiced until his retirement in 1944, specializing in surgery and gynecology.

Dr. Taylor was a member of the American Medical Association and the State Medical Association through Polk and Harris Counties Medical Societies. Elected to honorary membership in the State Medical Association in 1947, he was a former president of Polk County Medical Society and in 1930 served as president of Harris County Medical Society. A fellow of the American College of Surgeons and

a member of its governing board from 1941 to 1946, Dr. Taylor was a member of the South Texas District Medical Society, of which he was a former president, and a member of the Southern Medical Association and the Post Graduate Medical Assembly of South Texas. Dr. Taylor was formerly president of the staff and honorary consultant at Memorial Hospital and president of the staff and emeritus consultant of Jefferson Davis Hospital, both in Houston. He was also professor of clinical gynecology (honorary) at Baylor University College of Medicine, Houston. A thirty-second degree Mason, he was a past master of the Moscow and Camden lodges. Dr. Taylor was a member of the Presbyterian Church.

On March 1, 1893, in Moscow, Dr. Taylor married Miss Mattie Leggett, who survives. Other surviving relatives are three sons, M. J. Taylor, C. M. Taylor, D.D.S., and Judson Lucian Taylor, D.D.S.; two daughters, Miss Charlotte Taylor and Mrs. J. Stier Cunningham, all of Houston; a sister, Mrs. Jack Thomas, San Marcos; six grandchildren; and two great-grandchildren.

A. W. TERRELL, JR.

Dr. Alexander Watkins Terrell, Jr., Dallas, Texas, died at his home October 16, 1949, from cerebral hemorrhage.

The son of Mr. and Mrs. Alexander Watkins Terrell, Sr., he was born May 26, 1908, at Dalhart. He received his early education in the public schools of Fort Worth and was graduated with a bachelor of arts degree from the University of Texas, Austin, in June, 1930. After his graduation from the University of Pennsylvania School of Medicine in June, 1934, he served a two year internship from 1934 to 1936 and a one year residency in medicine in 1936 in the Hospital of the University of Pennsylvania, Philadelphia. He was the Edward Bok fellow in medicine in 1937 and the George Cox fellow in medicine in 1938-1939 at the University of Pennsylvania, serving as assistant instructor in medicine there from 1937 to 1939. Dr. Terrell practiced in Dallas from 1939 until his death.

DR. ALEX W. TERRELL, JR.

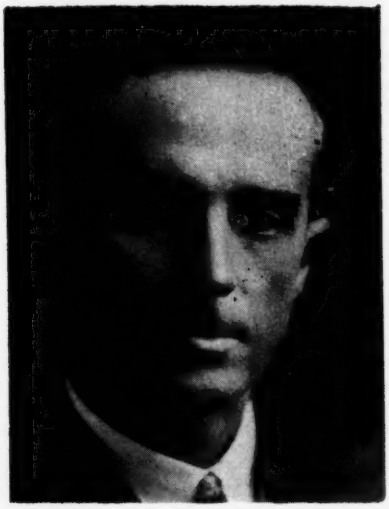
Throughout his professional career, Dr. Terrell was a member of the American Medical Association and the State Medical Association through Dallas County Medical Society. A fellow of the American College of Physicians, he was a member of the American Federation of Clinical Research, the Dallas Southern Clinical Society, the Dallas Academy of Internal Medicine, and the Dallas Internists Club. Dr. Terrell was affiliated with Alpha Omega Alpha, honorary medical fraternity, and Kappa Alpha and Alpha Mu Pi Omega fraternities. He was an instructor in medicine at Baylor University College of Medicine, Dallas, from 1939 to 1942, and an assistant professor from 1943 to 1945, and was associate professor from 1945 to 1949 at Southwestern Medical College, Dallas.

On December 6, 1941, in Oklahoma City, Dr. Terrell married Miss Jeanne Braniff, who preceded him in death January 7, 1948. His surviving relatives are his mother, Mrs. Alexander Watkins Terrell, Sr., Fort Worth, and two brothers, Joe C. Terrell, Fort Worth, and John L. Terrell, Dallas.

A. W. ROARK

Dr. Alfred William Roark, Saratoga, Texas, died in a Houston hospital September 12, 1949.

The son of William Charles and Susan (Langston) Roark, Dr. Roark was born February 7, 1882, in Kountze. Receiving his early education in the public schools of Hardin and Jasper Counties, he attended Washington University, St. Louis, and was graduated from the old Fort Worth School of Medicine. Dr. Roark began his practice as the company doctor for the McShane Lumber Company, Dearborn, Texas, from 1908 to 1912. He then moved to Saratoga, where he practiced until his death.



DR. ALFRED W. ROARK

Dr. Roark was throughout his professional career a member of the American Medical Association and the State Medical Association through Tarrant County, Jefferson County, and Hardin-Tyler Counties Medical Societies successively. A charter member of Hardin-Tyler Counties Medical Society, he served as its president in 1936. He was a former president and for many years a member of the Board of Trustees of the Saratoga Independent School District, and a member of the Christian Church.

In Kountze on February 21, 1909, Dr. Roark married Miss Rissie B. Collins, who survives. Also surviving are two daughters, Mrs. H. W. Cain, Houston, and Miss Mary Elizabeth Roark, Beaumont; a son, Alf Roark, Houston; and a brother, Milton Roark, El Paso.